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VETERINARY MEDICINE

SEPTEMBER 1937

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No. 9

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1959 East 72nd Place
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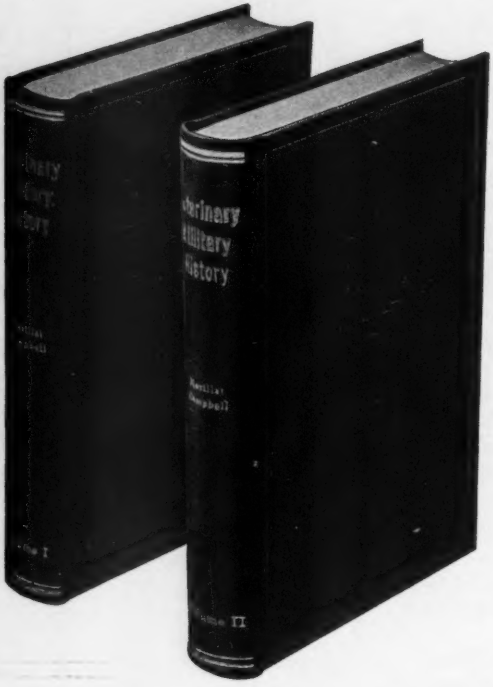
VETERINARIANS!

made this History;
Veterinarians wrote this History
All Veterinarians should read this History



Show me a veterinarian who does not understand that the accomplishments of his forebears should be enthusiastically and religiously preserved and I will mark him at once as a man of little worth himself.—*D. Arthur Hughes.*

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D. M. Campbell, Editor A. T. Kinsley, Associate Editor
H. J. Mostyn, 1265 Broadway, New York, Eastern Representative

Selenium in affected foodstuffs is present in the protein fraction. Little, if any, selenium is present as metallic selenium or in an inorganic salt.

* * * *

The monsters occurring in eggs produced on certain farms are not due to an inherited factor but are correlated with the toxic grains grown on such farms.

* * * *

The injection of selenium salts into the air cells of hen's eggs before incubation produces monsters similar to those occurring naturally. Sublethal concentrations of arsenic, fluorine, and lead ions produces ectopic conditions, but no monstrosities such as produced by selenium.

* * * *

The fetal guinea-pig is a suitable experimental animal for the demonstration of pathogenicity of members of the Actinomyces group. Smooth variants of Actinomyces are more likely to be virulent than those in the rough phase.

* * * *

It is a rather common belief that animals instinctively select foods most beneficial to them when a choice is offered. In an area where the forage possesses varying degrees of toxicity this instinct, if it actually exists which is doubtful, would have tremendous significance.

Long and Bliss report remarkable success in treating gas gangrene with sulfanilamide (prontosil). The action of this new chemical, against the Welch bacillus, being comparable to its effect on streptococci, pneumococci and gonococci.

* * * *

A dog named Abuwtiyuw, belonging to an Egyptian Pharaoh over 4500 years ago, was given royal burial at his death, according to an inscription, unearthed by the Harvard University-Boston Museum of Fine Arts expedition, exploring a cemetery near the pyramid of Cheops.

* * * *

From the tissue of the pancreas of the pig a substance has been isolated which inhibits the growth of the type of malignant cancer in chickens known as "chicken tumor I." The inhibiting action is definitely associated with the unsaturated fatty acid fraction of the pig pancreas.

* * * *

Determining the characteristics of streptococci obtained from freshly drawn milk samples, Plastridge and Hartsell at the Storrs experiment station found that either biochemical or serological tests alone may give satisfactory results. Both tests are necessary for the final identification of streptococci of bovine origin.

One of the signs of vitamin A deficiency is damage to mucous tissues such as line the inside of the eyelids.—*S. N. L.*

Vitamin C prepared from the wild rose is said to be more effective and to have a more pleasant taste than the vitamin C obtained from pine needles.—*S. N. L.*

Acne vulgaris is attributed to micro-organisims growing in the openings of the glands of the skin. There is little evidence to support the theory that diet is the cause of acne.—*S. N. L.*

Mucin which protects the lining of the stomach has for one portion of its molecule a substance called glycuronic acid. This acid helps the body to get rid of certain kinds of poisons by a chemical process of detoxification.—*S. N. L.*

Many viruses, in addition to requiring the environment within the cells for multiplication, have special predilection for nerve cells. An alum-picric acid spray has been designed to create a barrier through which the virus of anterior poliomyelitis cannot pass into the nerve cells.—*S. N. L.*

The proteins of our foods furnish us with the nitrogenous material which is necessary in building up our own tissues. To do this they must first be broken down into simpler products by our digestive secretions. The digestive acting upon them by means of the enzymes or ferments they contain.—*S. N. L.*

When chicks were fed for prolonged periods on a diet normal in all respects except for its complete lack of vitamin E, they all died, some of them suddenly, others after an illness. Examination after death disclosed the growth of new, cancer-like tissue which was destroying the normal tissue of liver, heart, pancreas, gizzard, spleen and lungs. The tumors belong in the class known as lymphoblastoma and appear very much like the tumors found in typical cases of leukemia in the fowl.—*S. N. L.*

Experiments by government scientists show that the gizzard of a chicken is a food grinder but not a digestive organ.—*S. N. L.*

One milligram of "heparin," a substance prepared from the livers of dogs, prevents clotting of about an ounce of blood for an hour. It has been used safely and successfully in human patients and dogs.—*S. N. L.*

When certain aniline derivatives are added to foods, the presence of vitamin B is indicated by a purple color of the test. Chief advantage of this method is the speed with which vitamin B determinations can be made.—*S. N. L.*

Egg white thoroughly mixed with potassium bromide and kept in an incubator at body temperature for several days yields a substance which makes blood clot quickly and firmly.—*S. N. L.*

A new rabies vaccine made from rabies virus cultivated in the laboratory protects mice against street virus and promises to give the same protection to dogs from the rabies virus which they may pick up from other rabies-infected animals. It is free from nervous tissue, and will not produce paralysis or any other untoward complication.—*S. N. L.*

Prontosil and prontylin do not kill the dangerous streptococcus germs, but check their growth so that they become ready prey to the disease-fighting white blood cells of the patient's body. Theoretically the chemical treatment should work in any disease caused by hemolytic stropticocci, including wound infections with streptococci.—*S. N. L.*

By using a high frequency electro-scalpel and coagulation unit, which seals the blood vessels as the tissues are cut, amputations may be performed with comparative safety. Coagulation of the tissues by electricity at the time of the amputation not only prevents hemorrhage but greatly reduces the clotting time of the patient's blood.—*S. N. L.*

Experiments conducted at the Rockefeller Institute for Medical Research lead to the conclusion that what have been called virus diseases are due to infection with just a few molecules of protein, and that a virus may be inactivated or made non-disease producing and yet retain its property of giving immunity to disease.—*S. N. L.*

* * * *

The human race devoured foods for how many thousands of years before any of us became endowed with sufficient curiosity to start a chemical investigation of them—not but what such efforts are laudable and valuable, but why starve to death waiting for complete knowledge? We haven't yet even mastered the chemistry of plain sugar metabolism. It was as late as 1920 that the molecular construction of our commonest carbohydrate, cane sugar, was first mapped (Irvine and Steele, *J. Chem. Soc.*, 117:1474, 1920.) And synthesis of it was not accomplished until 1928 (Pictet and Vogel, Comp. rend., 186:727, 1928). We still are in the dark as to how it is assimilated. If we have had to wait for a knowledge of sugar chemistry that long, is it reasonable to expect a complete knolwedge of vitamin chemistry before making use of them therapeutically?—*Vitamin News*, 5-96.

* * * *

In a review of our information about the animal reservoirs of the organisms of the Brucella group, before the 38th annual meeting of the Society of American Bacteriologists, W. A. Hagan, Dean, New York State Veterinary College, said it is regarded as adequately proved that human infections may and do occur as a result of contacts with cattle, goats and swine, and their secretions. There is little reliable information on the possibility that horses, sheep, dogs, and fowls may act as sources of infection for other species of animals and for man. While there seems to be no doubt that horses may sometimes act as such sources of infection, it seems highly improbable that sheep, dogs, and fowls ever play such a rôle, even though they may be invaded by Brucella organisms, and become reactors to the agglutination test for brucellosis.

Warming horses up whereby the membranes of the mouth and throat become dry and the saliva sticky, then feeding dry oats before watering, is one cause of choke. —*W. B. Redman.*

* * * *

Animal tests for vitamins should be used only to suggest deficiency syndromes, not to make standardization tests, because of the fact that many of the purified vitamins are specific for species.—*Vitamin News*, 5-95.

* * * *

Monetary loss incurred from the death of animals which perish of preventable diseases in China annually is conservatively estimated at $100,000,000.—*Insp. & Commerce*, Sept., 1936.

* * * *

In Mongolia, where animal-raising is the main occupation, 1,000,000 animals are reported to have died of preventable diseases in 1935, inflicting an economic damage of $50,000,000 to the Mongols.—*Insp. & Commerce*, Sept., 1936.

* * * *

Would Take Fur Farming Away From Bureau of Biological Survey

Largely because the fur industry is constantly growing, and because Wisconsin is one of the greatest fur-producing states in the union, Rep. Gerald J. Boileau of Wausau has introduced a bill to provide for classifying fur-bearing animals brought into or born in captivity as domestic animals or livestock.

The bill is backed by the American National Fox and Fur Breeders' Association.

Boileau's bill would amend several laws relating to domestic animals and would transfer jurisdiction over fur-bearing animals to the bureau of animal industry. It also would amend laws dealing with livestock diseases, quarantine districts, and exportation of diseased livestock, to include fur-bearing animals.

Boileau's secretary explained that similar legislation was enacted to bring poultry under the bureau of animal industry. Enactment of Boileau's bill would make possible federal experiments and disease control of fur-bearing animals as well as livestock and poultry.—*American Fur Breeder.*

Fundamentals of Livestock Management

Animal health is the foundation for profitable animal production. Which link in your chain is the weakest? Neglecting to provide these essentials for health decreases vitality.—*Col. of Agr., Univ. of Wisc.*

---

intravenously after toxemia has developed, but also where there is likelihood of generalized infection developing, a prophylactic intravenous injection of an antiseptic solution is prudent practice.

W. B. REDMAN.
Dow City, Iowa.

Injury to Vaginal Wall During Coitus

Recently, I received a call from the County Farm to see a small heifer that had been bred to the large, Holstein, herd bull.

The heifer was lying down, eyes staring, breathing accelerated and labored, temperature 104° F., straining with a vaginal discharge of blood and mucus. On examination, I found the penis had pierced the lateral wall of the vagina in two places during coitus. Expecting the development of peritonitis, I injected 7cc of a methelyne blue-nitrite compound intravenously and gave a laxative and two drams of tincture of aconite.

The vaginal tract was then flushed with a chlorine solution and the injured wall swabbed with tincture of iodine. Two days later this treatment was repeated and an antiseptic solution dispensed with instructions to syringe the vaginal tract once daily for seven days.

The heifer was back on her feed the fourth day, illustrating I think not only that it is good practice to administer antiseptics

* * * *

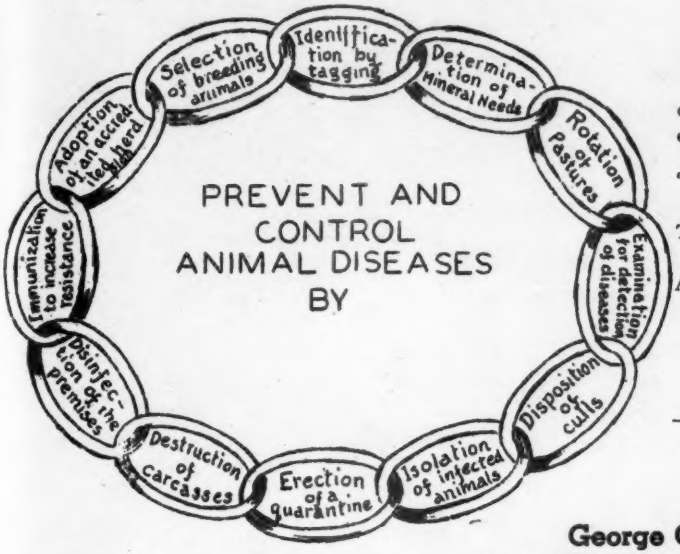
The veterinary profession must have an esthetic and cultural development to correspond to its professional attainments. The veterinary profession has unlimited possibilities. Nothing is more scientific or more of an art. Veterinary medicine is as sound as anything else in the world.—*M. C. Hall.*

* * * *

A Necropsy Indicated

A 1,400-pound gelding, 12 years old, had an enormous swelling on the breast, extending well up under the throat, back between the forelegs, back to the sheath and on the outside of each shoulder down to the knee. It was hard and feverish. Respiration was labored, the pulse fast, temperature 104° F., and appetite poor. The ailment started with a swelling in the left axilla, about six inches long and two inches thick and spread to the extent stated. I was called two weeks after the swelling was first noticed and could not get it under control. The horse lived another week and died. It was not a manger bruise. What caused it and what would have been the proper treatment?—*E. J. W., Ill.*

Principles of Livestock Sanitation



Animal disease is the chief limiting factor in successful animal husbandry.

"'For,' said the Deacon,
'it's mighty plain
That the weakest link
must stand the strain,
And the way to fix it,
as I maintain,
Is only jest
To make that link
as strong as the rest'."

—*Col. of Agr., Univ. of Wisc.*

Malpractice and Negligence

In former times it was the rule that, when a physician was sued for malpractice, the complainant must prove such negligence or lack of knowledge by the testimony of expert witnesses, in order to win a verdict.

Of late, however, the legal doctrine of *res ipsa loquitur* ("the thing speaks for itself") is being more and more employed in our courts, and when such a plea is sustained, expert witnesses are not required to prove negligence on the part of the defending physician.

The earliest examples of the use of this plea were probably in cases where a sponge or instrument was left in the body following an operation, but it is now being successfully employed in cases of burns from diathermy, x-rays, or hot-water bottles, the breaking of hypodermic needles in the tissues, etc.

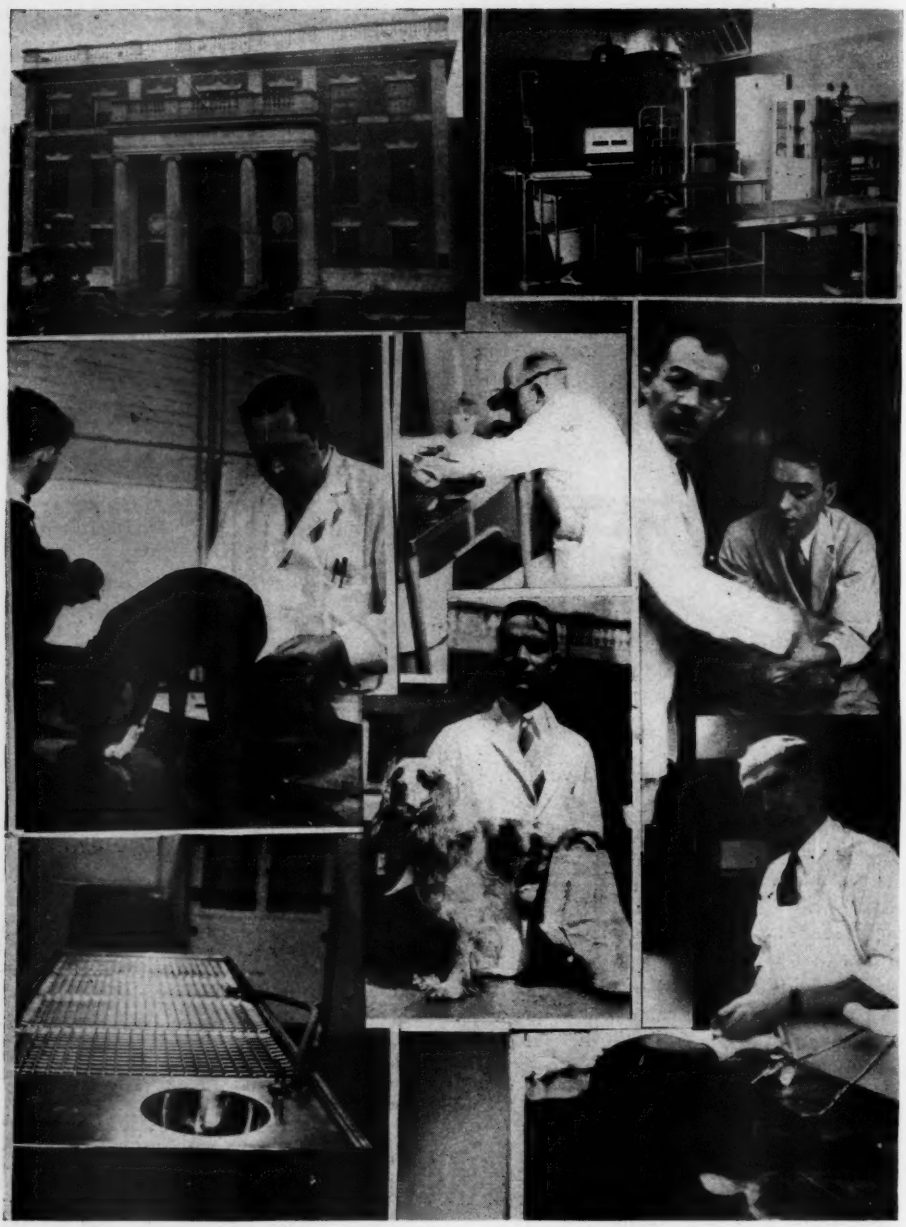
The increasing invocation of *res ipsa loquitur* as a rule of evidence, makes it incumbent upon all physicians in active practice to familiarize themselves with the circumstances in which it may be used, as a measure of self-protection.—*Clin. Med. Surg.*, 44:2.

George C. Faville

Doctor Faville is one of the older veterinarians in practice in this country. He was graduated from the Iowa State College in 1880, being the first veterinary graduate of that school. Since that time he has been uninterruptedly engaged in general practice, teaching, federal or state inspection; and is now practicing once more—57 active years of veterinary service. His present practice is a general one. Within ten days, he reduced a fracture in the leg of a canary and another in a cow. Both cases did well.

Doctor Faville is located in Emporia, Virginia, an average good farming country. His nearest confrere is 28 miles away. Without help, he supplies a complete veterinary service to his community, despite his four score years.

Doctor Faville achieved national prominence in the Veterinary profession early in his professional career. In March, 1884, a disease of cattle appeared in Coffey County, Kansas, which was pronounced foot-and-mouth disease by a local veterinarian. This diagnosis was confirmed by the state's veterinarian, by veterinarians sent to the scene by the U. S. Commissioner of Agriculture, the Canadian Department of Agriculture, and lay cattle commissioners of surrounding and even distant states. Doctor Faville, then State Veterinarian of Colorado, pronounced the outbreak ergotism, which diagnosis was later confirmed by D. E. Salmon and James Law and the excitement which was running very high in the cattle industry immediately subsided.



VIEWS OF THE ANGELL MEMORIAL ANIMAL HOSPITAL

Top: Front of the M.S.P.C.A. building at 170-184 Longwood Ave., Boston, which houses the offices of the Massachusetts Society for the Prevention of Cruelty to Animals, and the Angell Memorial Animal Hospital. The building was erected in 1914 at a cost of approximately $250,000; (2), the diagnostic laboratory (examination room in a less pretentious establishment) of the hospital. *(Continued at top of next page)*

Center: Dr. Hugh Frederic Dailey (U.P. '13) advising surgical treatment for luxation of the patella in a Boston terrier. Doctor Dailey has been with the M.S.P.C.A. since 1915, and is chief veterinarian since 1922. (2), Dr. C. Lawrence Blakely (U.P. '33) removing a foreign body from the digital pad. The dog is under nembutal anesthesia. Doctor Blakeley has been with the M.S.P.C.A. since graduation. (3), Dr. Rudolph H. Schneider (U.P. '15) obtaining a history of the case from the owner of the patient. Doctor Schneider has been with the M.S.P.C.A. since 1919; Assistant Chief Veterinarian since 1922.

Bottom: A specially constructed autopsy table with hot and cold water piping, electrically powered for complete elimination of odors. Approximate cost of table and its installation, $1,200. An electric motor operates a fan in the basement, which creates suction of the air from the table; this air is conducted through a shaft that extends above the top of the building. Autopsies may be made without the offensive odors usually present. (2), Dr. Todd O. Munson (U.P. '32) and a femoral fracture patient. Doctor Munson has been with the M.S.P.C.A. since 1933. (3), Dr. Gerry, B. Schnelle (U.P. '26) preparing for a reduction and fixation of a sub-trochanteric fracture of the femur in a four-year-old Boston terrier. The leg is being draped for the application of the splint (shown at the doctor's left) and tongs.

M. S. P. C. A. Animal Hospitals

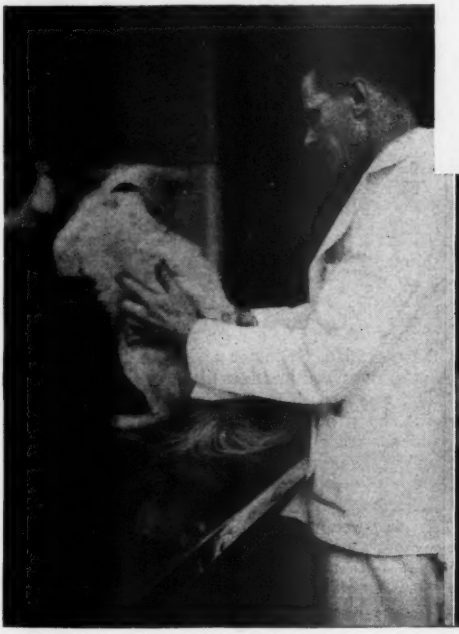
On this and the preceding page, views are shown of the Angell Memorial Animal Hospital of Boston, owned and operated by the Massachusetts Society for the Prevention of Cruelty to Animals. (The society operates an animal hospital in Springfield Massachusetts also). The work of this hospital has grown steadily since it's establishment in 1914. The annual report for 1936 shows that 8,774 cases were hospitalized and 24,701 treated in the clinic. Six full time veterinarians are employed.

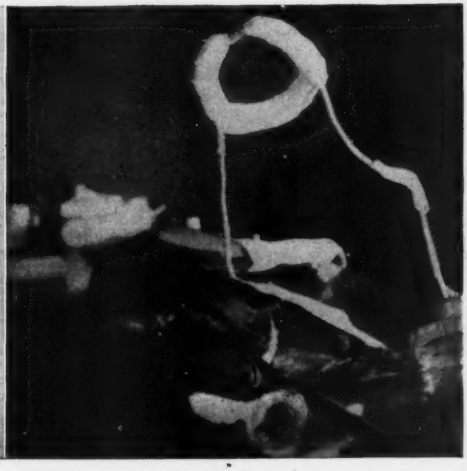
Fracture of the Femur in a Boston Terrier

The Boston terrier shown in the illustration was presented at the Angell Memorial Animal Hospital during the closing days of 1936 with a fracture of the upper third of the femur and treated by Dr. E. F. Schroeder. The Radiographs on the following page refer to this case.

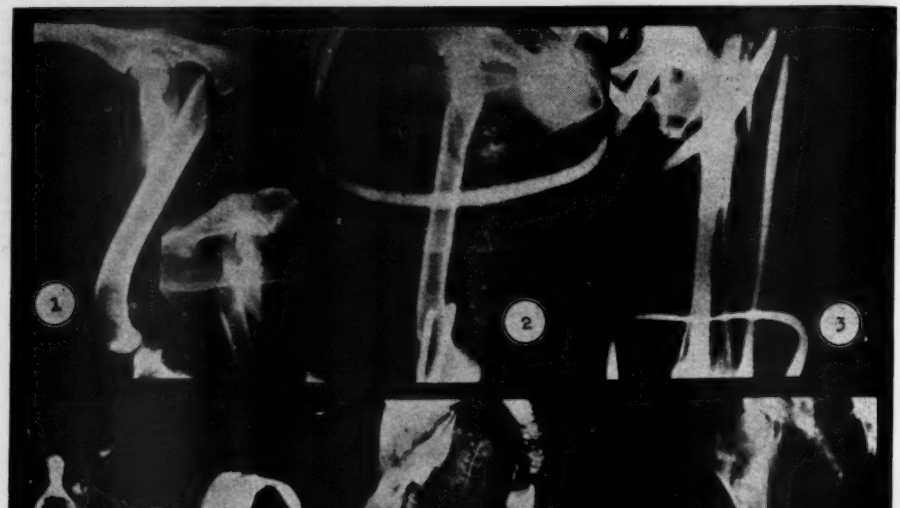
Explanation of Radiographs

Fig. 1. The fracture. Two views. The small view on close inspection will reveal a partial (incomplete) fracture of the femoral neck also. The radiograph is somewhat deceptive because it appears to show the fragments in contact in their displaced position. This was not so. The upper fragment was decidedly abducted and rotated outwardly. This can be visualized on noting the trochanter major in the radiograph. The thigh was greatly swollen as a result of considerable





Left: Dr. Erwin F. Schroeder (Texas A and M '05; O.S.U. '11) palpating for cystic calculi. Doctor Schroeder has been with M.S.C.P.A. since 1924. Below: A five-year-old Boston terrier with fracture of the upper third of the femur. The patient is under nembutal anesthesia, the leg has been prepared for splinting and the splint (also shown) has been wrapped with cotton preparatory to putting it on the leg.



Radiographs of a Fractured Femur in a Boston Terrier

hemorrhage due to the laceration of the muscles by the sharp fragments, and from the bone, due to the proximity of the fracture to the nutrient foramen. The sharp pointed proximal extremity of the lower fragment was adducted and pulled upward toward the tuber-ischium. The fragments were separated by interposed muscle. Reposition was impossible by the usual closed method of manipulation and traction. Open reduction was therefore necessary in order to free the fragments from this barrier of interposed muscle before reposition of the fragments was possible.

Fig. 2. Radiograph (lateral view) showing the fracture reduced. The line of incision was made on the lateral aspect of the thigh extending from the trochanter major to approximately an inch above the lateral condyle, through the skin and superficial fascia. A careful incision (longitudinal) through the aponeurosis of the tensor fascia lata exposing the anterior border of the biceps femoris. This was reflected posteriorly separating it from the quadriceps. By proceeding carefully need for invading muscle tissue was avoided and therefore unnecessary mutilation and danger of hemorrhage. In this manner the bone is readily exposed and approached.

Fig. 3. Showing an anterior posterior view of the reduction.

Fig. 4. A photograph of the patient and his splintage.

Fig. 5. A (close up) photograph to show the healed incision and the tongs in position. This photograph was taken on the eleventh day after the operation. Splintage and tong traction (*moderate*) was continued until the thirtieth day and then removed. The tong wounds appeared aseptic. The patient was discharged three days later and showed an inclination to bear weight on the limb. The owner reported a week later stating that the dog was using the limb cautiously. The patient was returned for examination at the end of the seventh week at which time he was walking quite well.

Fig. 6. Radiograph of the healed fracture at the end of the seventh week.

Receptivity and Resistance

The body plays a rôle no less important than the germs in the development of infectious diseases and their causes.[1] It can modify the soil which favors or retards the evolution of epizootics.

Alimentary insufficiency and over-feeding lower the resistance of the body and contribute to the development of infectious diseases and to the multiplicity of cases. This action is more marked in certain diseases than in others. Although lowered resistance of the alimentary type is difficult to prove with exactitude, the A-avitaminosis produced by the McCollum ration is scientific proof that animals can be made more susceptible to disease by alimentation. The seasonal outbreaks of hog cholera correspond to this type of alimentary depletion. Vitamin A and vitamin B are the only two of these factors, however, which have been proved to lower resistance, although there seems to exist seasonal modifications in the vital functions, noticed in growth, in changes of the composition of the blood and in the respiratory combustions which are perhaps not without relation to the appearance of epizootics at certain periods of the year.

It has been convenient to attribute the disappearance and return of epizootics to phenomena of immunity following outbreaks. In herds exposed for the first time all the animals are sensitive and contract the disease with the exception of those having resistance depending upon age, sex, breed or individual peculiarities. There are infections like foot-and-mouth disease which strike nearly all of the animals of a herd and others, like sheep pox which attacks only the sensitive at first but later as the disease evolves, the less sensitive also are stricken. Although they escape the first contact, as the virus becomes more abundant, the infection extends.

The epizootologic tableau is, however, not that simple. Owing to plurality of the virus (e.g. foot-and-mouth disease) the immunity is conferred only for one type without assuring against outbreaks from other types. Moreover, cured animals, immunized, conserve the virus in their bodies and remain capable of infecting exempt animals at any moment. The experiments conducted on mice with bacterial diseases show that while they may be immunized by one infection they remain capable of infecting their congeners. Artificial immunization has the same hazards as immunity acquired naturally.

The rôle of carriers which may be animals that have not been sick, or which have been sick and recovered, have primordial importance in epizootology as has been known long since.

[1] Panisset, Prof. L. 1937. Excerpt from *Recueil de Médecine Vétérinaire* 113:4.

* * * *

The Stages of Labor in the Bitch

The physiological course of labor in the bitch can be divided into three definite stages as in other animals. The first stage, the stage of dilation has no definite beginning, which we can recognize by clinical signs, except that the bitch tends to segregate herself, if kept under natural conditions. The uterine contractions exert pressure upon the membranes surrounding the fetus approaching the internal uterine opening and forces the bag backward into the thinned cervix, gradually dilating it.

The second stage is the stage of expulsion of the fetus. When the cervix is fully dilated and the membranes have ruptured, the abdominal muscles and diaphragm are brought into play along with contractions of the uterus, to force the fetus which has now become impacted in the pelvic inlet, through the vagina and vulva to the exterior.

The third stage is the stage of delivery of the membranes. This follows almost immediately after the expulsion of the fetus. The forces acting in this stage are the contractions of the uterus. A period of rest of varying length usually follows and then the same sequence of events is repeated, though now the first stage of labor is directed toward moving the next fetus to be born along the uterine horn to the pelvic inlet and not in dilating the cervix.—*W. McG. Mitchell* in *The Veterinary Record.*

Third International Congress of Comparative Pathology; An Interpretation of Its Significance

In April, 1936, the Third International Congress of Comparative Pathology met at Athens, the workshop of Æsculapius, Aristotle, Hippocrates and others who, 25 and more centuries ago, consolidated the legends and lore of still more ancient times into what was to become the cornerstone of the medical and veterinary professions of our day—two groupings which were harmoniously espoused in days remote, but completely divorced in Occidental nations during the Middle Ages, only to begin reviving their romance at the turn of the 20th century when Physician Bérillon and Veterinarian Lépinay of Paris were struck with the happy idea of founding the Society of Comparative Medicine which gave birth to the international congress above mentioned.

To be more precise, in 1902, Bérillon and Lépinay founded the Société de Pathologie Comparée which, ten years later organized an international congress under the same name—Comparative Pathology—with the aim of reëstablishing actual and spiritual coöperation between the medical sciences throughout the world. It is a long stretch between Æsculapius and Lépinay, but after all, time is a relative thing indeed in the march of man.

The Third International Congress of Comparative Pathology was not a dull, prosaic affair. It was opened by His Majesty, King George II, who reminded the congress of the importance of its undertakings and was addressed by His Honor Mayor Cotzias of Athens; Dr. G. Photinus, dean of the Faculty of the University of Athens, and President Bensis (Greece) of the congress. Fourteen countries were represented. Among the delegates were prominent figures well known in medical and veterinary circles. Of these the report contains such names as Feissinger, of the Medical Faculty of Paris; Nicolas, director of the veterinary schools of France; Prof. J. Russell Greig, of the Moredun Institute and the faculty of the veterinary school Edinburgh; Professors von Bergmann and Rossle, of Berlin; Prof. Rubay, director of veterinary education of Belgium; Prof. Huguenin, of Berne; Petit of the Pasteur Institute of Paris; Dufrency, plant pathologist of Bordeaux; Prof. Foex, director of the station of plant pathology of Versailles, to mention but a few members of the distinguished audience whose names will catch the eye of readers of scientific literature related to veterinary science.

In a brief address, published in the December, 1936, issue of *Revue de Pathologie Comparée*, the monthly spokesman of the society, Editor Lépinay, co-founder of the society and secretary of the French delegation, spoke reminiscently about the birth of the organization (1902) when, on account of ill-entertained prejudices, it was felt that the different groupings (physicians, veterinarians, pharmacists, phytopathologists) would adhere but timidly to such an organization. The long-forgotten past had to be reincarnated in a modern but then more thoughtful world. The international congress of 1936 in far-away Athens, which promises to reëstablish the ancient confederation of medical doctrines for the good of mankind, is the outward expression of the progress made toward that end in a short 36 years.

To what extent the movement, started by Physician Bérillon and Veterinarian Lépinay, has contributed toward the amalgamation of human and veterinary medicine in America, we leave for our readers to judge. The fact remains, however, that up to the turn of the nineteenth century coöperation between physician and veterinarian, abolished for 20 centuries throughout the world, did not exist in the United States. It has been only during the last few years (and slowly) that the medical profession has begun to turn some attention to its coöperation with veterinary medicine—a coöperation which died with the glories of Ancient Greece, its predecessors and its neighbors.

The ceremonies of the congress at Athens

were impressive, fascinating. The village of Æsculapius, named for the God of Health, the altar of Hygiea, the arena of Epidaurus, seemed to inspire the speakers by their solemn longevity and all they meant in the rehabilitation of medicine in our period. As one speaker declared, a meeting in Athens of scientists devoted to the study of medicine is "a pilgrimage of the spirit as well as the heart."

But what is most gratifying is that in 1902 veterinarians were first to lead in restoring the logical relationship between

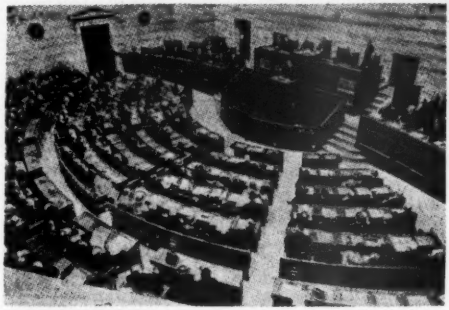


Fig. 1. The opening session of the Third International Congress of Comparative Pathology at Athens.

human and veterinary medicine, much in the same mood that prompted them to take the initiative in defending the teachings of Pasteur in whole-hearted fashion in the 1870's when the "wine chemist of Paris" was not being taken any too seriously by the academies of science.

As the modern world is gradually waking up to the fact that in the welfare of animals and plants lies man's only hope of health, wealth, comfort and the strength of his nation, it is quite natural that intelligent attention is being paid to comparative pathology in countries where hit or miss programs involving the application of the medical sciences are no longer tolerated, *ex necessitate rei.*

What is the matter with Greece, the one-time proud country, rich in beauty and culture, that it should have been practically erased from the society of nations? Leclainche, in his history of veterinary medicine, blames its decadence on Plato, who inculcated the notion that since animals have no soul they are not entitled to the blessings of (human) medicine. Whether this theory is rational or not, it is never-

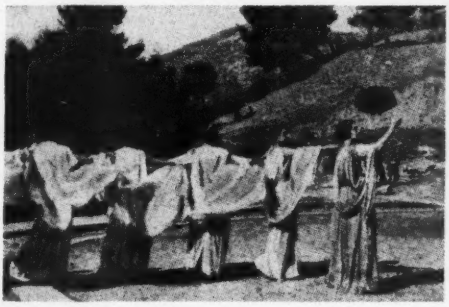


Fig. 3. One of the dances of the classical epoch of Greece reproduced for the session of the Third International Congress of Comparative Pathology at Epidaurus.

theless true that from that day forward Greece gradually went the way of the poor husbandman. Where the care of animals had been a veritable obsession, neglect of

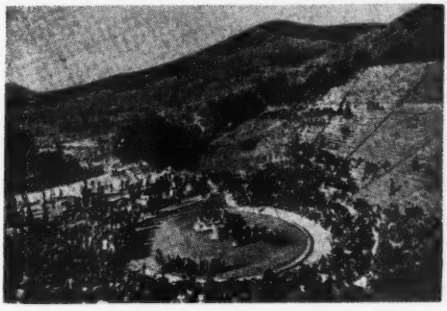


Fig. 2. A session of the Congress was held in the arena of Epidaurus.

them became a religion. As the street gamin would say, "Now look what ye done." The Third International Congress of Comparative Pathology, milling through ruins preserved by modern man, seeking to revive the wisdom once expounded in them, multiplies the magnitude of the tragedy. The moral may be read in every obstacle we place in the path of the veterinary service of our country.

Swine Balantidiasis

By J. D. RAY,
Omaha, Nebraska

The Author

PROTOZOAN diseases of man and animals are becoming more important as definite information is obtained about their nature and prevalence. Briefly, the parasitic protozoa are divided into four classes:—

1. Sarcodina, ameba in man and lower animals.
2. Mastigophora, flagelates, trichomonads, trypanosomes.
3. Sporozoa, coccidia, malaria parasites.
4. Infusoria, ciliates, *Balantidium coli.*

We are becoming more protozoan minded. The daily press has done much to inform the people about the prevalence and gravity of the amebic infections in man. Much information has been disseminated recently about coccidiosis in the various domestic animals and fowls. Trichomoniasis in fowls and cattle are recognized as important problems.

The ciliate, *Balantidium coli,* is recognized as an important pathogen of man. Swine harbor this parasite and, ordinarily, it produces no recognized lesions in this animal. In fact, many investigators say that it is a harmless entity. But the writer does not agree with this latter view. This opinion has been gained after numerous clinical observations of herds of swine affected with various types of enteritis. Many herds have been seen where *Balantidium coli* was considered to be the primary cause of trouble. They are associated with more or less definite symptoms and lesions and are easily recognized by microscopic examination of suspected material.

According to Walker[1], balantidia were first observed as parasites of the domesticated pig by Leuckart (1861 to 1863). They were found also by Stein (1862) and Ekecrantz (1869), Theobald Smith[2] (1910) considered the *Balantidium coli* a parasite of the normal intestine of the domestic pig and found that it multiplies rapidly and invades the tissues whenever opportunity is offered. However, he did not determine the ultimate effect of the presence of the parasite in the tissues. Walker[1], working with *Balantidium coli,* arrived at some very definite conclusions. He found that: "Balantidia are passed in the resistent encysted stage more or less constantly in the stools of parasitized pigs . . . *Balantidium coli* can produce bacteriologically sterile abscesses in the submucosa of an infected intestine . . . *Balantidium coli* is the primary etiological factor in the symptoms and lesions of balantidial dysentery . . . The latency prevalent in balantidiasis of man is due chiefly to the fact that the patient, although parasitized, is not infected with *Balanidium coli,* but in part to the chronicity of the ulcerative process in infected cases. . . . Every person parasitized with *Balantidium coli* is liable, sooner or later, to develop balantidial dysentery. . . . *Balantidium coli* var. *suis* is identical with *Balantidium coli* var. *hominis.* . . . The domesticated pig is the chief source of infection in the balantidiasis prevalent in the Philippine Islands."

In the writer's opinion there is just as much reason to think that swine may become infected by *Balantidium coli* present in the intestinal tract as in man.

The species of balantidia are hard to differentiate. Pigs harbor two types, the coli and suis. It has not been determined whether these are separate species. The suis type is found only in the pig, while the coli type is found in various animals. Hegner[3] found it "impossible to separate *Balantidium coli* of man and the coli type

from the pig by the size or shape, characteristics of the body and macronucleus." It seems to be unquestioned that human beings become infected, in many cases, with balantidia from pigs. It is difficult, if not impossible, to explain the occurrence of balantidiasis in man in any other way, except in certain localities where balantidia from monkeys may be the infective organism. Hegner[3] writes that pigs have been infected with balantidia from man by Brumpt (1909), Ohi (1923) and Andrews (1932).

Balantidium coli seems to be universally distributed wherever swine are found. Swine from some localities are more heavily parasitized than from others. A great number of microscopic examinations have been made of the content of the large intestines of swine from different parts of the middle west. *Balantidium coli* has not been found in all cases of intestinal involvement, but it has been found in every case examined where the ciliates were numerous, intestinal lesions were present and the consistency of the feces more or less soft. These observations are in accord with those of Pritze (1929), and referred to by Nelson[4]. Pritze found that heavier infestations of the organism were present in soft feces with a maximum content of undigested plants.

Much work has been done on the cultivation of *Balantidium coli* in artificial culture media. Media and methods have been developed that make this procedure practical. Jameson[5] and Rees[6] were among the early workers with cultures. A Ringer's solution is the base of such media. The formula used is:

Sodium chloride	6.50 grams
Potassium chloride	0.14 "
Calcium chloride	0.12 "
Sodium bicarbonate	0.20 "
Sodium biphosphate	0.01 "
Distilled water	1,000cc

Schumaker[7] used the following medium successfully in cultivating balantidia.

Sterile horse serum	3 parts
1% Asparagin in 0.65% saline	10 "
Ringer's solution	87 "
Sterile rice starch	small amount

This medium is titrated to pH 7.2 to 7.4 and cultures incubated at 37.5° C., for the best growth.

Schumaker found that *Balantidium coli* multiply equally well under either aerobic or anaerobic conditions. Deep or shallow media make no difference. The parasites settle to the bottom in either event. This led early workers to think they were anaerobic in nature. A temperature below 34° C. is unfavorable to growth. Originally it was necessary to transfer cultures every 48 hours. This is not necessary if a small amount of rice starch is added daily. They should be transferred once a week when starch is added daily, as the products of growth become concentrated to the point that growth ceases.

Atchley[8] made further additions to information on culturing balantidia. He found that 10% pig serum could be successfully substituted for the horse serum used by Schumaker. Atchley also developed a fecal extract medium that looks promising. It did not work as well as the Ringer's-serum medium, however, in growing single cell cultures[9]. He found that the larger volume of medium per individual makes for longer life and rapid multiplication. It was not necessary to sterilize the fecal extract medium used.

Glaser and Coria[10] found that initial transfers to artificial media were best obtained when a semi-solid medium was used. They modified Schumaker's medium as follows:—

Sterile Ringer's solution (50° C)	250cc
Standard 2% nutrient agar	25cc
Sterile rice starch	1 gram
Fresh sterile horse serum	12½cc

Mix and adjust to pH 7.2 to 7.4; tube into 15cc lots; set in ice box. Always warm to 37° C before use.

The reproduction of balantidia will not be discussed in detail. The trophozoites (vegetative forms) reproduce by transverse binary fission. A process of conjugation occurs in the life cycle at times. This is a complicated sexual process. Nelson[11], and others, have ably described it. Cysts are formed but little is known about these. They are the principal mode of transmission.

Balantidium coli (trophozoite) is an actively moving oval body about 60 to 100 microns long and 50 to 70 microns wide. It is covered with cilia and shows a funnel-shaped mouth at the anterior end; a bean-shaped macronucleus is distinct, and an oval micronucleus, two vacuoles and various sized granules. The size of the parasite varies with the stage being observed and the amount of food it contains. A rectal opening can be seen and frequently material is discharged and may appear to be dragging from the parasite. The balantidia are readily seen under the ordinary low power miscroscope and actively moving about if in fluid medium. Examine suspected material under a cover glass using only a small amount with a drop of water. The trophozoites disappear rapidly after death of the pig and sections of intestine submitted for laboratory examination may not show parasites when received at the laboratory, even though they were plentiful at death.

The incubation period in swine seems to be about 10 to 14 days depending somewhat on the severity of the infection. The usual history is exposure in sales pavillion, public yards, contaminated feed lots, or exposure to animals from such places. Controlled infections in experimental animals occur in one to four days[12].

Symptoms.—The animals gaunt up rapidly, with a rise of temperature and extreme weakness in many cases. Some individuals will thump due to weakness or the extreme toxemia. Diarrhea is usually present with whitish or yellowish flakes of exudate in a watery stool. Bad cases may pass considerable mucus which is frequently blood tinged. This may be mistaken for bloody dysentery. Death may occur in 24 to 48 hours after severe symptoms develop. The mortality may be high in heavily infected herds.

Lesions.—Ratcliffe[13] studied the intestinal lesions associated with balantidial infection in swine. He found, as most investigators have, that many swine carry balantidia in the intestine and are not affected by them; but, when young swine were infected by an organism of the Salmonella group, the ciliates invaded the intestinal tissues. He found that when ulcerations occurred in the large intestine of swine as a result of *Balantidium coli* infection, they were of a widespread superficial fashion. The protozoa apparently elaborate a cytotoxic or necrotizing substance that causes tissue degeneration. Lesions are invaded by intestinal bacteria. Therefore, the extension of tissue necrosis may be due to a combination of factors rather than to the parasites alone. The lymphoid tissues of the intestinal wall are important foci for extension of the disease to the submucosa.

Gross lesions as noted in the field vary. The mucosa is usually swollen and congested on the tips of the folds in the large intestine. There is no discoloration in the deep folds in the early stages or lightly infected cases. As the disease progresses, whitish or yellowish, fluffy, scab-like accumulations form over the congested areas of the mucosa. These have been termed "clabber milk-like" flakes of exudate and may be rubbed off easily. Microscopic examination of scrapings from these lesions or from the swollen mucosa usually reveal the *Balantidium coli* in great numbers. Bacterial infections in the lesions change their character rapidly and it is not uncommon to find more than one protozoon affecting the same animal. Such complications must be considered.

Various experimental animals have been used to study *Balantidium coli.* Guinea pigs are not satisfactory[14]. Rats proved susceptible[14,15] and much valuable information has been gained from their use. The parasites undergo division and conjugation in the rat, but do not produce cysts. Lesions are not produced in the intestine of the rat.

Treatment.—It was found that the diet of the rat largely determines its infectivity by *Balantidium coli*[14]. A heavy casein diet prevented infections. A high carbohydrate diet (85% or more) facilitated greatly the growth of balantidia in the intestine.

Schumaker[16] found that heavy infections with *Balantidium coli* in the domestic pig were accompanied generally by an in-

creased aciduric intestinal flora. Light infections with the protozoa were accompanied, in general, by a lesser number of aciduric organisms and more lactose fermenters and proteolytic anaerobes. Large amounts of starch were found in the cecum of pigs which had heavy infections with *Balantidium coli*. They were also associated with a diet high in carbohydrates in the form of grain.

Nelson[4] found that starch granules and blood cells were the only materials ingested and subsequently digested with any degree of favor by balantidia. Starch was preferable, indicating a definite selection in feeding by the parasite.

Gabaldon[17] found that the numerical population of *Balantidium coli* in artificially infected rats was not materially affected by increasing the viscosity of the intestinal contents.

Aguilar[18] treated forty cases of *Balantidium coli* infections in man by using repeated doses of stovarsol; but, he found it necessary to control the diet by reducing the carbohydrate consumed.

By accident, Greene and Scully[19], physicians, noted that a case of *Balantidium coli* infection in man that they had been treating for a long time with drugs and injections, with no benefit, improved and in a few weeks recoved completely on a milk diet. Since then they have treated several cases, all successfully, by giving them two and one-half quarts of milk daily.

Treating swine infected with *Balantidium coli* by administering medicinal agents has not been very satisfactory. Many agents have been tried on the parasites in vitro and most of those found to destroy the trophozoites were absorbed or changed, if given by mouth, before they reached the posterior bowel of the pig where the parasites are located. None of the preparations tested that are offered for sale for the treatment of enteritis in swine, had a detrimental effect on the balantidia, when exposed in vitro, in the dilutions recommended for administration to swine. Fluid extract of ipecac was tried and balantidia lived and remained motile in the full strength drug until it dried up on the slide —more than 30 minutes. Ipecac produced vomiting in pigs when given in medicinal doses and was not considered further. Ordinary concentrated lime and sulphur solution (dip), when diluted 1 to 100 destroyed balantidia in vitro. This solution was administered as a slop to infected animals and seemed to produce beneficial results. It nauseated some of the animals, but no other ill effects were noted.

A solution of 1 to 2,000, by volume, of formalin (37% commercial) in physiological salt solution killed balantidia in vitro. It was not possible to administer formalin by mouth and have it reach the posterior bowel and be effective. Rectal injections of a warm physiological salt solution containing 1 to 1,000 formalin were tried on a few pigs. This seemed to have a destructive effect on the parasites and did no noticeable harm to the pigs. Some of them that had been scouring and discharging many balantidia were voiding normal feces by the third day. A 70-pound pig will take from 1500 to 1800cc of a solution per rectum. An ordinary fountain syringe is used and the animals may be held head down or remain on their feet. The solution goes in better when the hind quarters are raised. It is possible by this procedure to fill the large intestine completely (to the tip of the cecum) with fluid. However, it does not pass the ileocecal valve.

Oil of chenopodium was found effective in vitro and seems to give good results in herds badly infected, but extreme care must be exercised in making a diagnosis. This is a drastic treatment for swine with enteritis and causes heavy losses if given to animals with acute infectious enteritis. The dosage of chenopodium used has been the same as for ascarids. This treatment has been enhanced greatly by a change of feed to increase the proteins and cut down on the carbohydrates. Until recently no attention was paid to diet in pigs infected with *Balantidium coli*. Consequently, swine became reinfected as soon as put back on the same feed, in the contaminated lots, regardless of the treatment used.

Since learning the effect of a milk diet on *Balantidium coli* infections in man and experimental animals, cases are recalled in herds of swine that had been treated with various internal medications, with temporary or no benefit, that were finally put on butter milk and immediately recovered. A milk diet has since been used with apparently good results in herds without other medication. This bids fair to become the logical treatment for these parasites and simplifies the procedure. But it must not be overlooked that butter milk is very irritating to, and contraindicated in some cases of acute enteritis in swine. For this reason it should be handled judiciously. After the animals have recovered the feed should be so controlled that the carbohydrate content will be below the danger line that promotes balantidial growth. This opens up another phase of the many nutritional problems that are constantly harrassing the live stock industry and baffling the veterinary profession. It appears that *Balantidium coli* infection in swine can be prevented by proper feeding.

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Relationship of Vitamin A to Reproduction

Inquiring into the reason for non-reproductivity in sows, Hughes[42] found that sterility resulted when the following diets were fed from the time the pigs weighed about 40 pounds: Barley and salt; barley, salt, and calcium carbonate; barley, salt, calcium carbonate, and casein.

Sows of similar weights, age, and breeding reproduced when fed: Barley, salt, calcium carbonate, and cod-liver oil; barley, salt, calcium carbonate, and chopped alfalfa hay; barley, salt, calcium carbonate, casein, and cod-liver oil; barley, salt, calcium carbonate, chopped alfalfa hay, tankage, and linseed meal.

Two lots of sows fed barley and salt for nearly a year did not reproduce, even when calcium carbonate was added. The sows in both these lots farrowed litters when cod-liver oil was given to one lot and when cod-liver oil and casein was given to the other.

Barley, when fed as the only source of vitamin A, did not contain enough of this vitamin for normal reproduction in the pig. Somewhat better results were obtained when an additional protein (casein) as well as additional vitamin A was added to a barley, salt, calcium carbonate diet. Pigs fed barley, salt, calcium carbonate, and casein had all the outward symptoms of a deficiency of vitamin A. In these studies, the addition of calcium carbonate to a barley and salt diet resulted in an increased appetite and rate of gain when fed to pigs.

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Restraint of Swine

By D. D. BAKER,
Wabash, Indiana

The Author

The restraint of animals is an art. For the student, it should be an important study. Successful restraint may mean a successful operation. Poor restraint is quite apt to result in a poor operation. Many practitioners have remarked that they needed better methods of restraint in their first years of practice. The clumsy, ineffective restraint used in learning different methods does not impress one's clientele favorably. It is prone to have the opposite effect.

Restraint is a hard subject to describe in print. To see it used at a clinic is the interesting way to learn it. At our clinic demonstrations throughout the country, the restraint of animals is one of the features attracting most interest.

In the restraint of swine, the problem is simplified by the size of the animals with which we have to deal, as well as by their restricted ability to inflict injury. They are not so highly sensitive to pain as some animals nor is there the need for so many operations on swine as are required in the horse or cow.

No operation on swine, nor on other animal, nor the restraint used, should be a display of torture. Therefore, anesthesia plays an important part in surgery. The most admirable restraint is complete anesthesia.

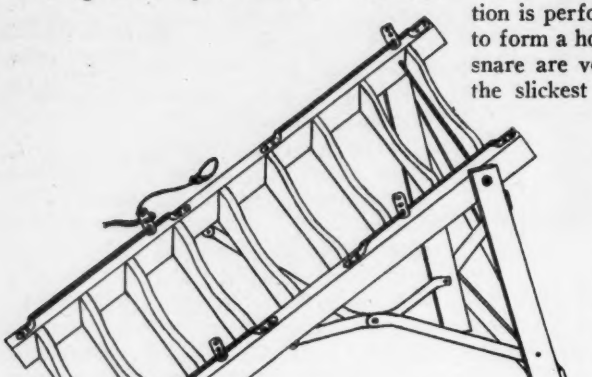
Restraint varies so much with the varying conditions that prevail on farms, that to lay down a set rule for all cases is impractical. Occasionally an owner will scratch a boar to get him to lie down, while he is vaccinated; again the dog is called in to tame an obstreperous sow that cannot be corralled, but will stand for anything while he chews on her ear. Some farmers will have a ringing crate suitable for vaccinating or castrating aged boars. So the problem of restraint is often solved by the owner. The position of restraint must be adaptable to the operation and often the owner's plan must be changed.

A trough of the V type, eight or ten feet long, is usually available on the farm. It provides a suitable operating table for ruptures and scirrhous cords. The trough should be inclined solidly against the wall or fence so that the animal may lie in it with its head down. This allows a dorsal recumbency on a 35° to 40° incline, and facilitates most abdominal operations. The feet may be stretched forward and backward and spread to the edges of the trough by using small ropes.

A ladder or panel gate may be used in a similar fashion. The open framework facilitates removal of scirrhous cords to better advantage than the trough. The traction on scirrhous cords should be backward and downward after the swollen cord in the inguinal region has been drawn out so that the openings in the panel or ladder will allow it to be pulled down over the tail. Cryptorchid pigs should be cast on the side for operation. For the inguinal operation, the same restraint is used as for rupture. In casting on the side, a rope should pass over the shoulders to prevent twisting or turning. The upper front leg should be drawn forward over the head and secured on the opposite side of the ladder from the lower front leg. Small ropes are quite essential in tying the legs of small shotes. Large ropes quite often slip off, no matter how tightly they are tied. The shotes may be anesthetized with a local anesthetic, with ether applied with a cone, with nembutal or with chloral hydrate. A wide margin of safety is, of course, desirable in using internal anesthetics, so that ether is often necessary to supplement the one adminis-

tered, but ether used alone with a cone and sponge proves very satisfactory.

A hog can be put in a state of basal anesthesia with chloral hydrate given with a stomach tube and pump, so that surgical anesthesia can be completed with ether or chloroform without any restraint.



Swine Operating Table

In passing the stomach tube on most shotes or mature swine, the animals should be snared and tied to a post. The assistant can then hold the speculum or mouth gag in place, while a tube is passed into the stomach and the solution pumped in. While other preparations are being made for operating, the narcotics take effect.

A very efficient operating table for swine for use in one's hospital can be built (see illustration). Following out the ladder idea for a table, the cross pieces may be placed closer together, the width should be made about 24 inches, the length seven feet, the heighth four feet. The legs can be made to fold to facilitate storing the table when not in use. Sliding lugs, as shown in the illustration, are convenient if added to the side rails to hold ropes for attaching the animals' legs. A great deal of time will be saved by using such a table.

In restraining boars for castration, a very simple method is to use a rope snare in the mouth; pass it around the right hind leg above the hock and draw the leg and head together until the animal falls over on the left side. The leg and head are secured in this position, while the operation is performed. A ring and loop of rope to form a hobble through which to pass the snare are very convenient. This is one of the slickest methods of restraint used in swine practice.

Anesthetizing aged boars for castrating was ably demonstrated by Doctor Downs at the A. V. M. A. clinic at Columbus. He snared and tied the animal short, wrapped a clean gunny sack around his nose and this was held in place by a loop of heavy rope drawn tight and secured back of the boar to prevent lunging forward. Chloroform was poured on the sack until complete anesthesia set in. About an ounce and a half was used in three or four applications. This makes a very humane demonstration and saves one's muscle where one cannot depend upon help.

* * * *

Effect of Ergot in Food

To determine the effect of ergot[48] upon the growth of swine, one paired feeding test was carried out in which the controls received a rye basal diet, and the experimental pigs received the same diet in which 1% of the rye had been replaced by ergot. From the results obtained there would seem to be little danger of ergotism occurring in swine that are fed grain containing ergot, because of their pronounced distaste for feeds infected with this fungus. There were no gross symptoms of ergotism in the experimental animals, but the ergot content retarded the growth to a statistically significant degree. Even when the ergot infected rye was stored for one year, it did not lose its distastefulness for swine.

Ergot develops not only upon the cultivated rye, but also on wheat grasses, meadow grasses, timothy, and several other grasses.

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External Parasites of Poultry*

By M. D. FARRAR, Urbana, Illinois,
Research Entomologist, University of Illinois

APPARENTLY the study of internal parasites has stolen the show in recent years from the study of external parasites. Dozens of papers have been written on internal parasites and very little on external parasites. This is not a reflection on the importance of the subject because external parasites, when present, will cut down egg production as surely as will internal parasites.

In 1933 a paper was published in Canada on the effect of flies in the poultry flock. This paper refers to a study of flies as they occurred in poultry yards, particularly the two largest groups—the blue bottle fly and the house fly. By capturing large numbers of these flies and feeding them to disease-free poultry they recovered coccidiosis organisms from both groups of flies. In other words, the birds which were fed the blue bottle flies acquired coccidia as did also the group fed house flies. This is of interest, as it illustrates one means of transmission of poultry parasites in the flock.

Interesting work has been done on fowl pox. In 1930 a paper was published in France on the transmission of fowl pox by mosquitoes. In this particular paper a certain species of mosquito was demonstrated to transmit fowl pox. Mosquitoes feeding on infected birds are able to transmit pox to healthy birds. Another experiment demonstrated that four different species of mosquitoes are capable of transmitting fowl pox to healthy birds. In the experiment these mosquitoes were able to transmit fowl pox for long periods after they had bitten infected birds; one species for 15 days, another 70, another 75 and the fourth for 210 days after it had bitten infected birds. They were able to isolate the virus from mosquitoes for as long as 185 days after they had fed on infected birds. Their conclusion was that these mosquitoes carried disease entirely mechanically; that is, the transmission was mechanical and not a condition which developed within the mosquito. They were also able to transmit viruses by means of stable flies and bed bugs, but not with poultry lice or mites. Their general conclusion on this particular study was that the transmission of fowl pox by insects as a whole is not very probable, although it is possible.

There is not a great deal new on the subject of insecticides for the control of external parasites. At the present time derris is coming into large use for many purposes in the control of parasites. The conclusion is that derris powder containing as much as 1% rotenone when dusted on birds gives adequate control. There is one objection, however. Rotenone is an alkaloid and tends to depreciate. The fresh material is fine, but the old is not so good. If the fresh material can be obtained I see no reason why derris powder cannot be used successfully in poultry parasite control.

Another development which has taken place is the use of nicotine sulphate to control poultry lice. The treatment is simple. The poultry operator likes it because it does not require handling. Merely paint a thin line of nicotine sulphate on the roosts and the parasites will be killed during the night. It is perfectly harmless if there is adequate ventilation. It controls lice nicely and is gaining wide use.

The question of the ordinary chicken mite in the hen house is one of sanitation. A well-cleaned poultry house where normal sanitary measures prevail will not harbor mites in sufficient numbers to be troublesome. The use of a number of other things, particularly crank case oil and products of that nature, give satisfactory results in the control of mites if they are used liberally on perches and hen house walls.

Another group of mites which does not occur very often is the feather mite. When the feather mite occurs in the flock, the

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birds become reddened by the mites crawling out on the feathers. The mite spends its entire life on the bird and control is entirely different. Fine flowers of sulphur and soap as a dip give satisfactory control. The formula is:

Finely powdered sulphur	2 ounces
Soap	1 ounce
Water	1 gallon

Another group of mites which attacks the skin is the scaly leg mite. These mites respond to the sulphur treatment mentioned for the feather mites. Immerse the feet and the fore part of the legs in the dip to kill mites.

For the control of lice on old birds the most practical treatment is sodium fluoride. It is very cheap and easy to use and is a standard product, easily obtained, and is the lowest cost treatment for the flock. It can be applied dry by the pinch method or the birds can be dipped in a solution. Either will give adequate control of lice. Nicotine sulphate treatment also is effective against lice. For young birds, both nicotine sulphate and sodium fluoride are rather toxic, and care is necessary in treating them. It is safer, though more laborious, to use the old treatment—greasing with vaseline and sulphur—on chicks.

Fried Bass

"One summer," began the Old Sheepman, "we ran our critters in a forest reserve not far from a lake that was just full of bass hanging around waiting to be caught. That was before the days of paved highways and autos, and a fish never saw a human being except a forest ranger or a big game hunter.

"Well, sir, we'd take a big, fat bass, weighing around five pounds, and carefully take out the entrails, and all the gray fat along the sides and back, and then skin him. We'd let him soak in salt water all night, and the next day cut him into square chunks; roll in cracker crumbs, and put the skillet in the oven. We'd pour the melted grease and butter over him, until he was a nice brown, and then put him on top of the stove to finish. By doing this there would be no muddy taste. With fried spuds; stewed tomatoes; sour dough bread; canned peaches and mountain coffee we managed to survive from one meal to the next."—*E. T. Baker.*

A Segmented Pig

March 5, 1937, a shote, weighing about 125 pounds, apparently cut around the circumference of the thorax, was brought to the veterinary clinic at Kansas State College.

The owner had purchased the pig, along with several others, at an auction sale seven weeks previously. At that time the pigs weighed 40 to 50 pounds. All were apparently normal. For a week, this pig had lagged behind the others and walked stiffly. The owner at first thought that the pig might have cut itself in the fence. This was disproved, because there were no holes in the fence and also there were no other evidences of scratches or abrasions. It was then that the owner decided to bring the pig into the clinic, thinking it must be some sort of a freak which might be of value for show purposes.

The wound was one-half to three-quarters of an inch deep around the entire body. Deep probing disclosed a binder-twine loop which was tied very securely.

Treatment consisted merely in cutting the binder-twine and removing it from the wound. The skin had not united over the twine, but the wound was so deep and the edges were in such close apposition that the twine was difficult to reach, even with a sharp pointed scissors. The wound was painted with tincture of iodine and the case dismissed.

R. R. Omohundro, K. S. C., '37.
Manhattan, Kans.

Maggot Infestation

Place 20 grains of methyl violet in a pint bottle; add liquor cresolis compound, two ounces; shake well, and fill bottle with kerosene. Apply as required.—*E. T. B.*

Stuttgart Disease and Nephritis*

By NEVILL S. KING, M.R.C.V.S.,
Leatherhead, England

FOR some time there has been a division of opinion as to whether Stuttgart disease and nephritis in the dog are one and the same disease or whether they are separate entities.

Stuttgart disease has been known by the following names: Canine typhus; hemorrhagic gastro-enteritis; dog plague; black tongue; la fièvre typhoïde.

There have been many suggested causes of this disease: anaphylaxis, intoxication, avitaminosis and infection with various organisms such as *Bacterium coli;* paratyphoid; spirochetes.

A move in the etiology was made when it was realized that the kidneys played an important part in the disease, and that a condition of uremia was frequently met with.

The very name "Stuttgart disease" seems to have been a stumbling block in the past, because it appears to be applied to several conditions the etiology of which may be quite different. Another difficulty is that these conditions have nephritis as their chief complication, and one has to decide whether this nephritis is of primary or of secondary origin. Again, the causes which are put forward vary with the countries in which the disease is said to exist. Thus, on the Continent, it is apparently recognized that spirochetes are the cause, while in America a disease known as black tongue[36] is said to exhibit symptoms similar to those of Stuttgart disease. The suggested cause of this is avitaminosis B_2. In this country opinion is divided—many suggesting that nephritis from any cause is Stuttgart disease, while others claim to separate the two. I include myself among those veterinarians who believe that what is known in this country as Stuttgart disease is, in actual fact, nephritis. I admit, however, that there may be an exception in what are described as "acute cases of Stuttgart disease," namely, acute hemorrhagic gastro-enteritis. Some of these cases are, without doubt, leptospiral in origin; others certainly are cases of acute uremia with terminal gastritis; while a third group may be classified as being a condition of anaphylaxis or as of unknown origin and without involvement of the kidneys.

In 1898[4] there appeared in Germany and other countries a disease which assumed the proportions of a plague. This was first described by Klett, who observed it in and around Stuttgart.

It seems reasonable to believe, in view of recent work carried out on the Continent, that this might have been the disease which is now known to be due to leptospiral infection, and to be closely related to Weil's disease in man. Stuttgart disease[36] is said to have been observed in America for the first time in 1887, and as far back as 1850 Hoffer described a disease of dogs under the name of typhus.

Nephritis

The etiology of nephritis, apart from possible avitaminosis B_2 and leptospiral infection, presents many problems. The chronic contracted kidney[3] which is so frequently met with at autopsy is so common a condition that one feels that there is still much work to be done both on preventive and curative lines. It is certain that variations in climatic conditions frequently play an important part in the onset of symptoms. One often finds a history of exposure to wet or cold immediately preceding an attack and many dogs develop the disease after a bath. Nephritis may be a result of injury, a legacy from distemper or other catarrhal fevers, or it may be a complication of them. Various organisms play their part in setting up the disease—amongst these may be found *Bacillus coli,* streptococci, and tubercle bacilli. Infection may be of the ascending or descending

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type.[5] Preputial catarrh, urethritis, cystitis may all be a cause of the ascending type, while prostatitis is considered by many to be a very frequent cause, the infection traveling up the ureters and setting up pyelonephritis. It is certain that an enlarged prostate gland and nephritis frequently go together. Ascending tuberculous nephritis secondary to primary lesions in the testicle or prostate is by no means uncommon.[6] Passage of the catheter may be a cause of ascending nephritis—the gum elastic type, mostly used in dogs, being difficult to sterilize. Descending nephritis takes place *via* the blood or lymph stream and may frequently be secondary to an infective pneumonia or endocarditis. Inhalation of certain poison gases can be included as a possible cause—nephritis being a common complication in humans affected with some poison gases. Absorption of toxins from the alimentary canal and certain chemical poisons may be responsible in some cases. One further cause may be mentioned, namely, arterio-sclerosis, either primary contracted type or secondary contracted type. Arterio-sclerosis is rare, but J. R. M. Innes[8] in 1930 recorded a case of each type. It is difficult to trace the origin of a lesion in the kidney, the glomeruli, tubules and interstitial tissue being so closely related that change in one must invariably affect the other.

Worm infestation must not be forgotten as a possible cause, particularly in dogs which have returned to this country from the East. A case was recorded by Henry Gray[1] in which *Eustrongylus gigas* or *Dioctophyme renale* was found in the kidney of a dog returned from India two years previously. This dog showed symptoms of nephritis. Referring to the presence of this worm, E. L. Taylor[2] states that it is found most commonly in the mink—usually occurring in the kidney but sometimes in the abdominal cavity. The length of time that elapsed before active symptoms were manifested in this case is interesting and it has been shown that some worms will remain *in situ* for several years. Renal calculi may also be responsible for setting up pyelonephritis—or even an acute uremia, by occlusion of the ureter. Chronic nephritis is frequently insidious.

Nephritis constitutes a very large proportion of the cases seen in canine practice in adult dogs—and its diagnosis is very important, not only from a curative point of view but from a preventive one also. One sees many cases of chronic nephritis which, had they been brought to the veterinarian soon enough, might have been avoided or at least alleviated. There are many cases of chronic nephritis that can be kept going for years by careful attention to diet, and these cases are numerous amongst older dogs. One wonders whether some of these apparently chronic nephritics may in fact be cases of nephrosis[38]—*i.e.*, cases where there has been an acute or sub-acute nephritis which have made a reasonable recovery without too much injury to renal tissue, but where there is always a leak of albumin. Several of this type have been seen, in which the specific gravity has gone up from a very low to a normal one, yet there is still a considerable amount of albumin present in the urine and the patient shows no other abnormality.

Symptoms.—One of the chief causes of sickness in the dog is disease of the kidneys. Frequently one sees a dog—the majority of such subjects are adult—in which the chief symptoms are frequent vomition, loss of appetite, and acute depression. This would suggest a possibility of acute nephritis, while a history of gradual loss of bodily condition, increased thirst and occasional bouts of sickness, in dogs past middle age, should lead one to suspect chronic nephritis. Many of these are termed "gastric" and have in the past been treated as such, but examination of a specimen of urine will enable a correct diagnosis to be made in both cases. The importance of urine examination cannot be emphasized too strongly, but it should be used as an aid to diagnosis only and the findings should be judged in conjunction with the clinical aspect. Nephritis may be divided into acute, sub-acute and chronic types.

The *acute type* may be seen in dogs of

both sexes; its onset is usually sudden, beginning with dullness, a disinclination to move about, inappetence, and increased thirst. Usually there is a rise in temperature, which may reach 106° F., in very acute cases. This falls in a few days until it becomes sub-normal, which may indicate the onset of uremia. An early symptom, which may not always be present, is pharyngitis—the back of the throat and mouth being a dirty color—accompanied by a slight cough. After two or three days profuse and frequent vomition occurs. Pain in the lumbar region is marked, particularly on manipulation, the dog often resenting even the slightest pressure. A "tucked-up" appearance and arched back, with a disinclination to walk, particularly upstairs, is seen frequently. There is often constipation at first but after the earlier symptoms diarrhea may supervene. Hematuria is sometimes present though by no means constant. Hepatitis may be a further complication[8], the mucous membranes of the eyes and mouth becoming icteric and bile salts and pigment being found in the urine. Dilatation of the sclerotic veins and injection of the conjunctivae is very marked. The mucous membrane of the mouth becomes red, particularly round the edges of the gums. Ulceration of the gums is common, especially round the canine teeth and inside the lips. A distinctive urinous odor is emitted from the mouth, due to retention of urea.

Uremia is a common sequel and is frequently met with at this stage. It is ushered in by convulsions, staggering gait, and incoördination of movement; coma sets in, and finally death occurs. Recovery will take place in many cases, but the acute type may develop into the sub-acute or chronic.

Sub-acute nephritis is less severe than the acute and symptoms are not so marked. It may be a "flare up" of the chronic type. Occasional vomition and thirst are seen. Loss of bodily condition is not so great as in the acute form. Temperature is raised only one or two degrees, and pain in the loins is not so marked. The tongue has a dirty brown appearance. Ulceration of the mouth is not so frequent but the mucous membrane is inflamed at first and later becomes pale in color. The stale urinous odor is not so noticeable. Skin lesions in the form of dry scurfy patches may be present[6]. Sub-acute nephritis frequently develops into the chronic form.

Chronic nephritis is most commonly met with in dogs past middle age and more especially in the male, though it is seen in the bitch. Its onset is slow and it may be a number of weeks before any very marked symptoms are seen; in fact, many cases are so indefinite that it is impossible to make a diagnosis without the aid of urine analysis. Early cases show only an increased thirst with occasional sickness—the vomition often occurring in the mornings only, and the vomit rarely containing food. It consists of white froth and usually follows the drinking of water. As the disease progresses the dog has such a crav ing for water that he fills the stomach persistently until there is a "pot-bellied" appearance. Appetite becomes variable and there is a slow progressive loss of bodily condition. The temporal and masseter muscles frequently become atrophied. The visible mucous membranes become pallid, and there is a distinct, almost characteristic, odor from the mouth; ulceration of the mouth and inside the lips is common. The gums are sore and bleed when touched.

Owing to the increased work placed on the heart, hypertrophy is common and dilatation of the left ventricle is produced, with not infrequently a systolic murmur due to mitral regurgitation. This is evidenced in the pulse[6] which is hard and bounding, and quite inconsistent with what one would expect to find in a dog whose condition would suggest a weak pulse. Edema is not common but it does occur in a few cases where the affection of the heart is very marked. Glaucoma, according to McCunn,[14] is frequently seen in conjunction with kidney disease and any such case will merit further investigation. Change of temperament is in some cases one of the first symptoms to be noticed;

dogs which were formerly placid becoming irritable, bad tempered and nervous. Eczema, either in the form of moist patches, or as a dry, scurfy condition—both of which are extremely resistant to treatment—is a frequent complication. History of stiffness and an inability to jump on chairs, or the suggestion that a dog suffers from rheumatism, should make one immediately suspicious of possible kidney disease, particularly if the dog be past middle age.

Fits, either in the nature of epileptic attacks or hysterical barking, and rushing about, are often one of the first symptoms observed; indeed, they may develop twelve months before other symptoms show themselves. Here again urine analysis will prove valuable in distinguishing these fits from others, such as those seen in true hysteria. Chronic cases ultimately terminate in death from uremia. As in acute cases, there is a more marked desire for water, a sub-normal temperature, complete loss of appetite, profuse vomiting, incoordination, paralysis, coma and death.

Diagnosis.—This may be confirmed by examinations of specimens of urine, though the testing of one specimen only is apt to be very misleading.

In nephritis of any type there are marked variations in the composition of urine, and albumin is almost always present in some degree. In acute nephritis the specific gravity is usually raised and albumin may be present in large quantities. The reaction of urine in both acute and chronic cases is usually acid, but occasionally it may be neutral.

In chronic cases, the quantity of albumin gradually decreases and at the same time the amount of urea is diminished in the urine and is raised in the blood. This is due to damage to renal tissue and shows that the kidneys are unable to excrete urea. Urine of chronic nephritis gives a persistently low specific gravity which may drop to 1003.

The passage of urine at any time with a urea content of less than 1.5% is uncommon, while if it is less than 0.75% the outlook is serious[8,9].

The urea concentration test is impossible in most cases as it requires the collection of urine at regular intervals, and passage of the catheter is not desirable.

Space will not permit the detailing of urine tests, most of which, with the exception of bacteriological examination which may involve cultures, can and should be carried out by the practitioner himself. Most of the tests are easily performed and have been dealt with very ably by Professor Boddie in a previous paper[6, 9].

The testing of samples of blood will also be of considerable value, though the withdrawal of from 5 to 10cc is not always easy to carry out in the dog, but a test of the urea content of the blood will be of material help—the amount of urea varying from 50 milligrammes per cent in the normal animal to over 400 milligrammes per cent in acute cases[7].

The use of the ophthalmoscope will be of service in cases in which the eyes are involved[14].

A further method to establish the extent of damage to renal tissue and the rate of excretion is the use of x-ray. Pyelography, though expensive, may prove to be of great value in this direction. It consists of the intravenous injection of uroselectan "B," and the subsequent taking of pictures at intervals after the injection. The rapidity with which the uroselectan is excreted by the kidneys is an indication of the amount of damage to renal tissues. This method is employed with success in human diagnosis. The number of cases in which we (King, Uvarov and Withers) have practiced this method has not yet been sufficient to justify the expression of opinion as to its effectiveness in the dog.

Stuttgart Disease

The definition given of this disease in this country in Woolridge's Encyclopedia[11] is: "A disease of dogs characterized by hemorrhagic enteritis, with a great tendency to necrosis of the mucous membrane of the mouth, tongue and alimentary tract."

Acute, sub-acute and chronic types are recognized.

The acute form is said to occur in dogs

that have a previous history of gastric catarrh, and in old dogs that suffer from nephritis. It occurs most frequently in dogs past middle age and may appear as an epizoötic or enzoötic.

Woolridge states that the feeding of healthy dogs with discharges from affected animals or portions of the stomach or intestines has frequently failed to produce the disease, though there are said to be cases on record where canine typhus has been set up. Transmission by lice or fleas has also been suspected by Woolridge[11]—organisms, corresponding to those found in lesions of the alimentary tract, being demonstrated in such insects.

In the *acute form* onset is sudden and may prove fatal in twenty-four hours. There is refusal of food, vomiting and great depression. The vomit is at first greenish in color, but later is blood-stained and has a very offensive odor. Temperature is at first high, but later drops below normal. There is an increased thirst and the patient vomits almost immediately after taking either solid or liquid. Loss of bodily condition is rapid and very marked. Anemia and dehydration soon follow. The patient becomes weak and has a "tucked-up" appearance. Often there is an arched back. In the cases that live for several days there is extensive ulceration of the mouth, which first appears inside the upper lips and on the gums and sometimes on the tip of the tongue. The mouth is cold and clammy, and a catarrhal coating is deposited on the teeth, dark brown in color, becoming blood-stained and extremely offensive; there is frequently dribbling of cold saliva. The patient becomes prostrate, and coma supervenes a few hours before death.

The sub-acute form usually begins with congestion of the pharynx and is accompanied by a slight cough. There is occasional vomiting and the appetite may be retained for a few days. Thirst is noticeable and vomition becomes more frequent. The dog exhibits signs of pain in the abdomen and may cry out if picked up. The eyes become sunken. Constipation, in the early stages, is followed by diarrhea, which is often blood-stained and offensive. Ulceration of the mouth and dribbling of saliva are marked. The tip of the tongue becomes congested at first, but later turns brown and frequently becomes necrosed at the tip. The pulse becomes fast and weak. Temperature is raised only two or three degrees. Loss of condition becomes pronounced and the patient may finally die in a coma. Tenesmus is frequently seen, due to irritation of the lower part of the bowel.

The chronic form is met with occasionally. The symptoms consist of persistent vomiting of a yellowish viscid substance, irregular bowels, great thirst and progressive emaciation. The mucous membranes are anemic and the temperature is normal or sub-normal. Occasionally mouth lesions, similar to those seen in the acute form, develop.

These symptoms as described by Woolridge appear to be almost identical with those of nephritis.

Leptospiral Disease—Spirochetosis

For some years investigators on the Continent have associated Stuttgart disease with leptospiral infection, and have noted the presence of spirochetes (*Leptospira ictero-haemorrhagiae*) in Stuttgart disease, which is associated with an acute nephritis in addition to the characteristic hemorrhagic gastro-enteritis[20]. Buccal and lingual ulceration is not seen in every case of leptospirosis[28]. Lukes[18] in 1922, demonstrated the presence of leptospira and at the same time he informed the Brunner Biological Society that he had succeeded in proving systematically the existence of spirochetes in Stuttgart disease. Since that time much work has been done by Wirth, Klarenbeek, Voet, Schüffner, Kotter, Schultz and others. In 1925, Okell and Dalling[10] showed that leptospiral infection was a cause of infectious jaundice in the dog. The importance of these discoveries is far reaching. There seems little doubt that leptospiral jaundice and Stuttgart disease are closely related, many cases of Stuttgart disease being actually leptospiral jaundice, the icterus of which is absent. There is another aspect which is important

to both veterinarians and physicians—that is the close relationship of leptospiral jaundice in dogs to Weil's disease in man. The organism is carried by the brown rat and infection takes place by contamination of water or food material by urine from an infected rat—leptospira frequently being found in the liver and kidneys. Cases of humans being infected from dogs must be considered possible[22]—a serious position, if true, for the veterinary surgeon and one demanding early and correct diagnosis. How far leptospiral infection is responsible for kidney disease in the dog it is difficult to estimate, but many dogs in Germany, Austria and the Netherlands are said to show signs of acute, sub-acute or chronic nephritis together with the presence of spirochetes either in the urine or blood. Spirochetes have also been demonstrated in sections of the kidneys[16]. They may persist in the urine for some considerable time after recovery has taken place, probably at least a month, so that these cases are a very definite source of spread. Stuttgart disease and leptospiral jaundice appear to have a seasonal incidence which in the two diseases is similar. Old records from the Vienna Veterinary College (based on post-mortem returns) show that the disease is more prevalent during the summer months. The interconnection between the two diseases is further demonstrated by the clinical course and by the blood picture. There is a rise in the red blood corpuscle count and hemaglobin content, a leucocytosis, mostly affecting the neutrophiles, polychromasia and an increase in blood platelets.

Okell, Dalling and Pugh describe two types of leptospiral jaundice, hyperacute and icteric, due to *Leptospira ictero-haemorrhagiae,* but more recent investigations abroad have established the existence of another leptospira—*L. canicola.* Again, *Leptospira canicola* has been shown to be the cause in 90% of the cases in which the nephritic symptoms have predominated[15]. This organism, unlike *L. ictero-haemorrhagiae,* the organism responsible for leptospiral jaundice and Weil's disease, does not cause jaundice[13] and according to reports is not found in the brown rat.

Some interesting experiments have been carried out in conjunction with the two diseases. Wirth[26] found spirochetes in the kidneys of 14 cases of leptospiral jaundice. Kidney suspensions from two cases transmitted the disease to puppies after subcutaneous inoculation. Similar suspensions from cases of Stuttgart disease were inoculated into seven puppies—four of which developed leptospiral jaundice. Spirochetes were seen in the kidneys of these puppies after death. The infection was passaged several times in other puppies. Puppies which were inoculated developed the disease in 10 to 18 days and died at 19 to 23 days. He also produced the disase in puppies by the subcutaneous inoculation or ingestion of urine from wild rats taken from a canal in Vienna. Leptospiral infection has been met with fairly frequently in the Netherlands[21] and though both *L. ictero-haemorrhagiae* and *L. canicola* were found, *L. canicola* was much the more common one. Identification was confirmed either by isolation of the organism or more frequently by serological tests.

Diagnosis of leptospiral infection is not always easy; the organism may be found in the urine or blood, its presence in either depending on the stage which the disease has reached. In the later stages, *i.e.,* when the disease is approaching the chronic stage, many of the symptoms will have disappeared and one will have to rely almost entirely on examination of the urine for the presence of the organism. Leptospira may be found in the blood at the third to the fifth day and in the urine from the seventh to the tenth day[12]. Their numbers may vary in the urine and there may be few or many. In cases where the urine is acid there may be very few—the acid hindering or preventing the secretion of live leptospira[12]. This is, perhaps, an important factor in the treatment. Diagnosis[22] depends on the demonstration of the organism by direct or indirect means:—

1. The examination of urine or blood by dark field illumination.

2. The measurement of the lytic power of the blood for leptospira.

3. Agglutination tests (in two cases re-

ported the blood showed agglutinins for a number of strains).

4. Culture of the organism in special media.

5. Guinea pig inoculation—leptospira being subsequently found in the blood of the inoculated animal.

Guinea-pig inoculation is not always successful—a difficulty being found in effecting the first passage in *L. canicola* infection.

Symptoms of Leptospiral Disease.—There is a rapid febrile course, extreme prostration and depression, complete loss of appetite and great thirst. Often there is passage of blood-stained, mucus-coated feces, vomition of bad-smelling material, hemorrhages from the lips, gums and nose. Dyspnea frequently occurs with a short cough. Death may take place in a few hours, or the dog may live for several days. Icteric symptoms are met with only occasionally. Urine is always dark colored and passed in small amounts. Albuminuria is invariably present. Specific gravity falls to 1005[18]; the reaction is always acid; renal cells, some leucocytes and sometimes casts are present. The quantity of blood urea is much increased. Post-morten examination shows very marked lesions—a hemorrhagic gastro-enteritis—the contents of the intestine and sometimes of the stomach being bloody and chocolate colored. There are ulcers in the buccal mucous membrane, and alterations in the renal tissue— an acute interstitial nephritis being present. Although nephritis is almost always a constant complication it is not of primary importance in post-morten diagnosis[24]. Icterus may be absent[28]. The cardiac endothelium, particularly on the left side, degenerates, and this is followed by desquamation and a tendency to thrombus formation. The myocardium shows diffuse hemorrhages and infiltration. Chronic interstial nephritis is a common sequel in those cases that do not prove fatal in the acute stage. Chronic cases show similar lesions which are older and may have undergone calcification. Some cases may show lesions of both acute and chronic types.

Black Tongue

This disease is seen in America and is said to be allied to Stuttgart disease.

Recent experiments have shown this to be due to a deficiency in vitamin B_2[29], and to have produced a condition resembling pellagra in humans[35].

In 1907, Chittenden found that dogs deprived of meat and milk fell ill with salivation, stomatitis and diarrhea, which is so characteristic of the condition known as black tongue, and met with most commonly in the Southern States of North America. Certainly the disease picture in naturally occurring cases is very similar to that of nephritis. Cases are described as being of two types—those with and those without nervous symptoms together with skin lesions. Nervous symptoms which manifest themselves by convulsions of the chronic type invariably prove fatal.

Age, sex and breed appear to have no noticeable influence on susceptibility[36], though in a report from one clinic it is interesting to note that hounds, pointers, setters and collies from suburban and rural districts constituted 85% of the patients treated, and that their average age was 3½ years.

All experimental attempts at artificial transmission have failed and all efforts to make cultures from heart blood have been negative[31].

Blood and urine analysis of specimens from cases revealed an increase in non-protein nitrogen, urea nitrogen, uric acid, and a fall in the chloride content of the blood. Urine was high in nitrogen and low in chlorides and contained albumin and casts.

H. C. Rea[36] records the treatment of 255 cases which included yeast, sodium chloride and glucose, lime water and calcium gluconate, carotene extracts and glycerine, and liver extracts. Liver extracts with sodium chloride appeared to give the best results and feeding of raw beef and canned salmon was said to be beneficial.

There are several points that seem to stand out in this disease. First, the similarity of symptoms to those of nephritis, together with the blood and urine reports.

Secondly, the fact that at one clinic the majority of cases were those of sporting dogs, *i.e.*, dogs most likely to be subjected to exposure to weather conditions, such as wet and cold (a frequent contributory cause of nephritis). Thirdly, the treatment recommended was similar to that which is adopted for nephritis.

One is inclined to ask "Is this a true avitaminosis with secondary nephritis, or is it nephritis with secondary avitaminosis?" A combination of the two is possible and it has been suggested that a deficiency in vitamin B_2 may be purely a secondary one due to faulty absorption—often a result of protracted gastrointestinal disturbance, such as is seen in nephritis[32].

The experimentally produced black tongue seems to differ from the naturally occurring cases, and much more closely resembles human pellagra.

Work on this subject has been done by Chittenden, Goldberger, Wheeler, Blackstock and others. Rhoads[34] and Miller[37] in 1932 succeeded in producing experimentally a condition of chronic black tongue with anemia, by means of a diet almost free from vitamin B_2. Control animals which were given four grams daily of liver extract together with the same diet remained in perfect health throughout, and examination of their blood failed to show any significant variation.

Interesting changes in the mouth were noted—particularly of the tongue. There was anorexia and diarrhea and often blood was passed. Loss of weight was striking—being about 20% of the original.

The general results obtained were characteristic of the macrocytic anemia of sprue and pernicious anemia in man[34]. The prophylactic and therapeutic properties of foods rich in vitamin B_2 were very marked. Yeast, milk, and liver extracts are rich in vitamin B_2[33].

Treatment.—Warmth and attention to diet are essential. Foods rich in protein should be withheld. Barley water should be given instead of water and limited in quantity. Milk, cream, glucose, honey, Benger's Food, etc., are all good, while injections of glucose saline and calcium gluconate will be found particularly helpful in cases of dehydration and collapse.

Bread and butter, and milk puddings may be given after the acute stages when vomition has ceased, and fish, poultry and boiled mutton may be given in small amounts as the patient is convalescing. Alkalis such as sodium bicarbonate will prove useful and combinations of bismuth will be of value in checking vomition. Salicylates are useful in relieving pain. Attention should be paid to the heart by the administration of digitalis, nux vomica, caffein and camphor in oil and ether. Nervous symptoms may be controlled by sedatives and respiratory changes in uremia may be helped by the injection of calcium gluconate. Rectal injections of normal saline solution may alleviate straining when hemorrhagic enteritis is present.

Mandelic acid[30] has been used as a urinary antiseptic in the treatment of nephritis, but it is suggested that it should not be employed when the renal function is impaired. Intravenous injection of formalin has also been used[8].

In leptospiral infections a polyvalent antispirochete serum has been used with success. Kantorowicz[23] records seven cases in which serum was given in Stuttgart disease and appeared to be of curative value. Various other medicinal aids have been used in leptospiral infections with variable results. Amongst these are pallicid, introcid given intravenously and salvarsan[17]. Rudolf and Mendal[27] report 100% success with magnesium perhydrol administered frequently and in small doses.

Conclusions

Nephritis, from various causes, is a common condition in this country. [England].

Hemorrhagic gastro-enteritis is frequently leptospiral in origin.

Black tongue in America appears to be associated with kidney disease, though in the experimentally produced condition the kidneys do not appear to be involved.

"Stuttgart disease" is a name that has been applied to all three conditions.

Nephritis is undoubtedly a common complication of the condition known as Stutt-

gart disease in this country, and is frequently seen in leptospirosis on the Continent.

Leptospirosis (producing acute hemorrhagic gastro-enteritis) is known as Stuttgart disease abroad.

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Manual removal of the entire placental membrane if possible, flushing womb with a hot, mild, saline solution, spells no discharge plus a good fee and a well satisfied client.—*W. B. Redman.*

Horse & Mule Association Pulling Contest

The National Horse Pulling Contest will be held at Columbus, Ohio, this year in conjunction with the National Percheron Show and National Dairy Show, October 11-16, 1937. The contest has been officially designated as the National for 1937, by the Horse and Mule Association of America, whose rules and regulations control all official pulling contests held throughout the world.

Cash premiums aggregate $1700, of this, $200 will be paid on Monday, October 11th, to local teams. Pairs under 3000 pounds weight, (horses to be under 16½ hands height, 66 inches) will compete for $800; preliminaries will be held October 12 and 13, and all prizes except the 1st, 2nd and 3rd and 4th will be settled on those days: top four teams will compete on Saturday, October 16, to settle final rankings. The pairs 3000 pounds and over will compete for $700; preliminaries will be held October 14 and 15 and the highest four pairs will be held over till Saturday, October 16, for the finals.

All contests — preliminary and finals — will be held on the clay floor in the live stock judging coliseum on the Ohio State Fair grounds, Columbus, Ohio, between 1 and 5 each afternoon, exact hours to be announced later. Teams winning in the preliminaries, which have to be held over for finals on Saturday, will be fed and stabled as guests of the National Dairy show from the time they win in the preliminaries, until the finals are over.

In addition to the National several hundred local pulling contests will be held this season under the auspices of the Horse and Mule Association. For details or for directions for putting on an official contest in your locality write, Wayne Dinsmore, Secretary, 407 South Dearborn St., Chicago.

Stricture of the Esophagus in a Cow

A Jersey cow, about six years old, vomited up her food in two or three hours after eating, plastering the walls of her stall with the contents of her stomach. She continued this for two or three weeks, then weakened and died. Her food consisted of ground corn, wheat, and pea hay. I would like to know the cause and the proper treatment.—E. J. W., Ill.

Comment: The first case I ever saw of the kind described was in a yearling white-faced steer. It would graze on the fresh green grass until it would partly fill the rumen and then rumination would start; it would eject one cud after another until there would be as much as a peck of cuds on the ground before it. The steer was sent to slaughter and I followed him through for autopsy, and found that some time prior he had swallowed some sharp object that cut a slit in the mucous membrane of the lower part of the esophagus, and a fibrous proliferation of the fascia had involved the left vagus nerve.

Case No. 2, a Jersey cow with the same symptoms, and autopsy the same.

Case No. 3, was sent to market; no autopsy.

Case No. 4, a two-year-old Jersey bull with the same symptoms as shown by the other three. Diagnosis according to autopsy on the first two. But I passed a stomach tube, and when it contacted the obstruction and I tried to push it down, the bull nearly tore up the barn and came near dying from suffocation as he quit breathing for awhile. Of course I quickly drew the tube out when the bull started to throw such a fit. When the bull finally got on his feet again, the owner said pass it again, and I did, and when it came to the object I put all the force on the tube that I had and shoved the tube into the stomach. The animal threw another fit and came near passing out, but didn't. The bull didn't eat anything for many days but drank water. Finally he started to eat, at first bran, later other food; but it was weeks before he ate normally. He looked like a skeleton for several more weeks, but made a complete recovery, and has shown no evidence of stomach trouble since.—S. L. STEWART.

* * * *

According to mink breeders, the gestation period of the mink varies from 40 to 69 days. Female mink rear litters numbering up to 10.

* * * *

ILLINOIS VETERINARIAN

On the opposite page are halftones of 64 of the members and visitors who attended the annual meeting of the Illinois Veterinary Medical Association at Springfield, Feb. 18-19, 1937. The names and addresses (left to right) are:

Top row: L. A. Shaffer, Mt. Carmel; Robt. Gysel, Chicago; D. E. Sisk, Mansfield; W. A. Young, Chicago; C. L. Miller, Oak Park; C. A. Dionne, Kankakee; P. T. Fehrenbacker, Philo; R. J. Sutter, Waukegan.

Second row: E. B. Ingmand, Indianapolis, Ind.; J. D. Ray, Omaha, Nebr.; J. S. Koen, Chicago; H. H. Seely, Jerseyville; H. E. Biester, Ames, Ia.; C. C. Hastings, Williamsville; S. L. Stewart, Olathe, Kans.; A. Prader, Carbondale.

Third row: J. E. Kelberg, Princeton; R. E. Ouderkirk, Reynolds; O. E. Lummis, Paloma; H. C. Rinehart, Springfield; W. M. Head, Walnut; F. O. Whiteman, Carrolton; R. O. Mudd, Biggsville; J. D. Shute, Easton.

Fourth row: C. H. Nyes, Cambridge; R. C. Calkins, Rock Island; O. L. Campbell, Astoria; W. C. Scotten, Monmouth; H. C. Pugh, New Windsor; W. O. Hilyard, Little York; H. L. Moser, Pontiac; R. J. Newman, Kenny.

Fifth row: Mr. C. Holmes, Peoria; R. O. Nye, Buda; C. F. Pauley, Kirkwood; John D. Reardon, Kansas City, Kans.; J. V. Lacroix, Evanston; E. E. Byerrum, Warrenville; C. W. McLaughlin, Melvin; J. O. Head, Carlisle.

Sixth row: Mr. J. S. Koen; F. W. Lupfer, Galva; J. W. Lucas, Abington; H. R. Simpkins, Elmwood; F. E. Brown, Blandinsville; W. E. Miller, Galesburg; A. E. Dickerson, Springfield; F. A. Walter, Lemont.

Seventh row: O. B. Hess, Fostoria, Ohio; J. H. Crawford, Hinckley; H. C. Wadleigh, Seaton; L. M. Darst, Princeton; Earl R. Kennedy, Moline; L. A. Merillat, Chicago; R. M. Carter, Alexis; L. Finney, Georgetown.

Eighth row: G. E. Hunt, Champaign; R. C. Julien, Indianapolis, Ind.; E. Wilham, Mt. Pulaski; Fred Graves, Belvidere; D. A. Cahill, Springfield; L. V. Puckett, Mt. Vernon; T. P. Gallahue, Waukegan; F. C Yabsley, Cissna.

Water Hemlock Poisoning

Poisoning of cattle by water hemlock has often been called parsnip poisoning due to the fact that the two plants resemble one another. Wild parsnip may be given in large quantities with no ill effects, but just a few roots of water hemlock will poison an animal.

Both the water hemlock and parsnip plants vary in height from two to six feet and have leaves very much alike. The water hemlock has hollow stems, many branches, and numerous clusters of small white flowers. The parsnip flowers are bright yellow. The parsnip has long, white tapering roots growing deep into the ground, while the water hemlock has many branched roots growing close to the surface. The water hemlock has a disagreeable, nauseous odor which becomes very strong when the leaves or stems are bruised. This affords a sure quick means of distinguishing between the two plants. Both plants grow chiefly on low-lying marshy ground, along creeks, and ditch banks, ripening in August and September. Poisoning occurs early in the spring or late fall. The root is the poisonous part of the plant.

Symptoms.—The history is that the cattle have been turned out to pasture a short while and the owner finds one or more dead or in convulsions. The first symptoms that will be noticed, if the poisoned animal is seen in time, is a wild staring, twitching, or popping expression of the eyes, nostrils dilated, and the neck arched. This is followed by salivation which may be slight or hanging from the mouth in long foamy shreds. Bloating is now noticeable. The animal trembles slightly, urinates and defecates spasmodically, and runs a short distance before falling and going into violent convulsions which remind one of the effects of strychnine. In the last stage the animal may live from a few minutes to an hour.

Treatment.—In view of the fact that because of the convulsions the stomach cannot be emptied the logical thing to do is to attempt to control the convulsions physiologically by giving chloral hydrate, or nembutal (pentobarbital sodium) and chloral hydrate. If noticed in the first stages chloral hydrate as a drench is sufficient. If upon arriving the animal is in violent convulsions drenching is impossible, therefore nembutal, acting more rapidly than chloral hydrate, is given intravenously. It is followed by chloral hydrate, given intravenously or per os, as the convulsions will stop immediately upon giving nembutal. Nembutal alone is not sufficient because of its short hypnotic period, therefore it is necessary to follow with chloral hydrate.

The above treatments have been carried out successfully in a number of cases.

EMIL E. GRINSTEAD.

Yakima, Wash.

Water Hemlock (*Cicuta maculata*)
The spindle-shaped and lower stem, the leaves, flowers and fruit are shown three-eighths natural size; also a cross-section of seed X4. The water hemlock is an extremely poisonous plant—U. S. Dept. of Agr.

* * * *

There are 15 million dogs in the United States, according to an estimate [polite term for guess] and over two million of them are pedigreed.—*S. N. L.*, 32:845.

* * * *

A new, rapid, inexpensive test for pregnancy (in women) has been devised by Hubermann. It consists in the intradermal injection of certain pregnancy hormones. In nearly 500 trials it was 90% accurate in positive and 95% accurate in negative cases.

Osteomalacia in a Jersey Cow

A grade Jersey cow, six years old, had dropped her fourth calf at full term on March 10, 1937. Her third calf had been aborted at about seven months, in the opinion of the owner, due to a kick by a horse. This cow has passed two tuberculin tests, the last about January 1, 1937, but has not been tested for Bang's disease.

The cow cleaned normally at calving. Afterwards, however, she did not act normally as to appetite or milk production. About April 1, there appeared thickened areas in the skin over the entire body, varying in diameter from ¼ to 3 inches, painful on pressure. There was no pus or break in the skin. The head from eyes to nostrils appeared widened, with skin quite firm and adhering to the bony structures. The hind legs, below hocks, became edematous; the front legs were not affected. The skin, except the head, was quite loose. The temperature was 102.6°F.; pulse 102; respiration 24; the gait slow. There was no edema in the region of the brisket; though, the skin was thickened along the throat. The feces were about normal. The condition continued to get progressively worse until the cow had lost nearly 150 pounds in weight.

Evaluating the symptoms given, it was evident that there existed a disorder of metabolism, as indicated by the loss of appetite, falling off of milk production, and the emaciation. The diseases of metabolism that come to mind are parturient acetonemia and osteomalacia. The enlargement of the bones of the face, and the bony nodules all over the body, together with the thickening of the skin suggest osteomalacia. The edema mentioned was not in conflict with the symptom complex of this disease; although, from a differential point of view one would have to consider puerperal sepsis and traumatic pericarditis.

The subsequent course and symptoms seem to rule out these latter assumptions. A mineral supplement was administered in the feed, after which considerable improvement in condition could be noticed from week to week.

E. C. HEINSEN.

Riga, Michigan.

Anesthetization of Chimpanzees*

Barbiturate anesthetics are widely used by veterinarians for general anesthesia in small animals, but they are less frequently employed with the larger forms. The use of these drugs with adult chimpanzees at the Yale Laboratories of Primate Biology, Orange Park, Florida, began as a result of a search for satisfactory methods of removing infants from their mothers. During the first few months of life, while the baby clings constantly to its mother, any attempt at forceful separation is extremely hazardous and impractical.

Because the chimpanzee is particularly wary of any attempts to disguise drugs in milk, fruit juice, or solid foods, efforts in this direction were soon abandoned. The subjects readily coöperate, however, in the procedure of rectal injection, after a brief period of training. Nembutal and sodium amytal have been used repeatedly in this way during the past two years with satisfactory results. Complete anesthesia usually is obtained from nembutal within a few minutes.

Twenty-seven cases in which nembutal was used, in doses ranging from 11 to 51.8 milligrams per kilogram of body weight, indicate that the minimum anesthetic dose for adult chimpanzees in good health is 25 to 30 milligrams per kilogram. Younger animals may require from 30 to 40 milligrams per kilogram.

It is possible that the rectal anesthesia method may prove useful with other large animals. Its application would depend almost entirely upon the ability of a given subject to respond to the training. In the chimpanzee the problem of training is almost negligible.

J. H. ELDER,

Yale Laboratories of Primate Biology.
Orange Park, Fla.

* A partial summary of an article entitled "Methods of anesthetizing chimpanzees" which appeared in The Journal of Pharmacology and Experimental Therapeutics, Vol. 60, June, 1937.

Vitamins and Distemper

VI CANINE BLACKTONGUE

W. O. FROHRING, Cleveland, Ohio

WHILE considerable confusion exists in the minds of many in regard to the vitamin B_2 or G complex, this is particularly true regarding the blacktongue preventing factor. Since the albino rat is used universally for test purposes in the biological assay of material, the fact that the rat either does not require this factor or synthesizes it, may possibly be an explanation of the confusion in connection with this nutritional factor necessary in the nutrition of the dog.

During the last decade, Goldberger and his associates [1-2] have demonstrated and described a clinical picture of blacktongue in dogs and pellagra in man and have presented evidence which warrants the assumption that both diseases are caused by a lack of the same factor whch is a component of the vitamin B_2 or G complex.

This work stimulated a great deal of research in the study of human pellagra and the interest in canine blacktongue consequently became a secondary issue. A review of the literature reveals only a few scattered reports on experimental canine blacktongue. Rhoads and Miller [3] produced experimental canine blacktongue in dogs using a diet similar to that of Goldberger[1]. They found that the disease is often acute and rapidly fatal, but in a certain number of the dogs, a chronic form was produced which featured macrocytic anemia with low hemoglobin and red cell count, loss of weight, diarrhea and atrophic glossitis. The symptoms were prevented by the addition to the diet of four grams of liver extract daily, and were cured by administration of materials rich in the vitamin B_2 complex. It was therefore concluded that the most probable etiology of the disease is the lack of some substance closely associated with what had been formerly called vitamin B_2 or G.

Wheeler & Sebrell [4] of the U. S. Public Health Service describe clinical and postmortem findings in dogs used in the dietary investigation of blacktongue, the special feature of which was a yellow, friable, fatty liver. The clinical features of the disease included chronic ulcers of the chest and legs, vomiting and bloody feces, with lethargy, lowered resistance and coma before death. "Yellow liver" is much slower in development than blacktongue, and it is suggested that it may be caused by long standing partial deficiency of the blacktongue preventing factor.

Crane-Lillie and Rhoads [5] demonstrated the neural pathology of blacktongue and showed the resemblance to the changes noted in human pellagra.

Birch and György [6] in a large series of experiments on dogs showed that neither flavin (riboflavin) nor vitamin B_6 would cure the lesions developed on the blacktongue producing diet, but that yeast and liver extract were good sources of the blacktongue preventive factor. The results of Birch and György were confirmed by Koehn and Elvehjem [7].

FROM this resume of the significant contributions to our knowledge of canine blacktongue it is evident that the symptomatology and clinical picture of the disease are multiple and varied.

Canine blacktongue has not, as yet, been produced in young puppies and only one investigator (György) has attempted to produce the symptoms of blacktongue in animals fed on a synthetic diet. Therefore, in this report there are presented observations made on several puppies on Goldberger's diet and on a synthetic blacktongue producing diet.

Experiment with the Synthetic Blacktongue Producing Diet

A litter of two mongrel puppies of a medium-sized breed was used in this experiment. The puppies were approxi-

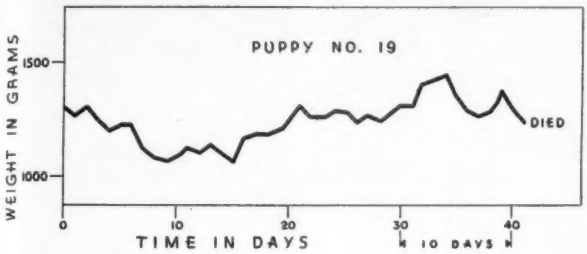
mately six weeks of age and had just been weaned.

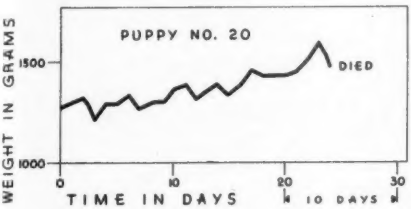
Diet.—Puppies 19 and 20, each weighing about 1kg were given a synthetic blacktongue producing diet of the following composition:

Synthetic vitamin-free bitches' milk, as reported by Frohring,[8] plus daily supplements of crystalline vitamin B_1 (10 units per kilo), lactoflavin (30 gamma per kilo), and vitamin B_6 (50 rat units daily) to each puppy. (The vitamin B_6 was given as cooked white maize meal and was mixed with the synthetic vitamin-free bitches' milk and fed to the dogs. The vitamin B_1 and lactoflavin were injected subcutaneously daily. The puppies were also given 2cc of cod liver oil daily.

Observations.—The puppies took this bitches' milk very readily and their weight remained constant, not varying more than 200 grams during the first 20 days. On the 24th day, puppy No. 20 did not eat and died the following day. Autopsy findings were not significant and no symptoms of the deficiency were noted. Puppy 19 showed a slight gain in weight during the next week and died on the 43rd day. No deficiency symptoms could be observed. Figures 38 and 39 show the weight curves of puppies 19 and 20.

Summary.—Two young puppies given a synthetic diet consisting of vitamin-free bitches' milk, supplemented with vitamin B_1, lactoflavin and B_6 failed to gain weight and died on the 25th and 43rd day of the experiment, respectively. No deficiency symptoms were noted at any time during the experiment.





Figs. 38 & 39. Chart showing the result of feeding a synthetic vitamin-free milk supplemented with adequate vitamin A, vitamin B_1, lactoflavin, vitamin B_6 and vitamin D. This diet contains none of the blacktongue-preventing factor. It is evident that one or more factors are missing.

Experiments in Which the Goldberger Blacktongue Producing Diet Was Used

Diet.—Puppies OA and OB, weighing about 7 and 6kgs, respectively, were placed on György's modification of the Goldberger diet which is as follows:

White maize meal	600
Dried pea meal	75
Extracted casein	90
Cod liver oil	28
Cottonseed oil	45
$CaCO_3$	45
NaCl	15

The white maize meal, pea meal, and casein were cooked in a double boiler for two hours, after which the other ingredients were added. The diet was given *ad libitum*. In addition, the dogs were given a daily subcutaneous injection of 80 international units of pure crystalline vitamin B_1.

Observations.—The puppies lost weight gradually and became emaciated and inactive. After 50 days on this diet, no mouth lesions were apparent. At this time (53rd day) there was observed a redness of the mucosa of the cheeks and flora of mouth. After the 55th day, the diet of dog OB was supplemented with 10 grams of Flavex* daily. This failed to improve the

* A crude dried form of lactoflavin derived from milk.

dog's condition, either in appearance or weight. On the 59th day, puppy OB showed a marked diarrhea with bloody feces. The mouth was very inflamed and the tongue pale—almost a grayish white. The animal was now in a moribund condition and injection of liver extract failed to bring about recovery. The animal died shortly afterward. At autopsy, the lungs were bloody and congested. Smears and cultures from the lungs and pleural cavity yielded a pure culture of *beta* streptococcus. Necrotic areas were noted along the inner surfaces of the lower lip.

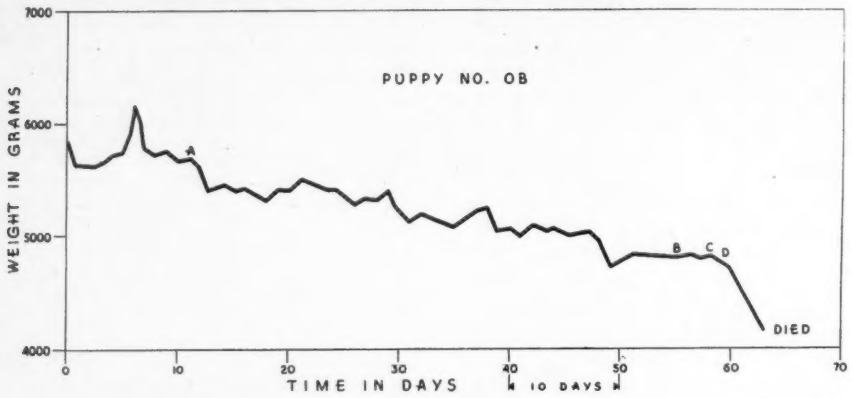


Fig. 40
"A"—Vitamin B_1 injections begun. 10 units per kilo injected subcutaneously daily.
"B"—Redness of mucosa of cheeks and flora of mouth.
"C"—10 grams of Flavex daily.
"D"—Liver extract—4 grams.
"E"—Diarrhea—very bloody feces—mouth inflamed.

Puppy OA was given 500 gamma lactoflavin subcutaneously on the 60th day. The animal's condition failed to improve, and shortly after this, bloody diarrhea with mucus in the feces was noted. The dog showed no improvement, when liver extract therapy was attempted, and death occurred within a short time. The tongue showed atrophy around the edges and deep transverse ridges across the upper surface, while the entire mouth was severely inflamed and hemorrhagic. The weight curves and time of appearance of the various conditions are illustrated in Figs. 40 and 41.

Summary.—Two puppies developed early symptoms of blacktongue, i.e., diarrhea, mouth lesions, etc., on the modified Goldberger diet when given adequate vitamin B_1 and lactoflavin supplements. Parenteral liver extract late in the course of the deficiency failed to improve the animals' condition, probably because of the infection contracted in the interim.

EARLIER in this series of experiments I reported results obtained in omitting the entire B_2 complex from the diet of puppies, using the synthetic bitches' milk. A larger number of puppies was used in these experiments than was used in the blacktongue diet just mentioned.

IN reviewing the observations that were made upon these puppies deprived of the entire B_2 complex, the rapidity with which diarrhea developed strongly suggests that in most of those puppies a deficiency of the blacktongue factor was one of the first to manifest itself. In two particular instances a bloody diarrhea developed. These observations in connection with blacktongue suggest interesting practical application in veterinary practice by the use of substances rich in the blacktongue-preventing factor. Chronic gastroenteritis in older dogs offers interesting possibilities in therapy in connection with the blacktongue preventing factor. The

profound effect of the absence of the blacktongue factor on the entire gastro-intestinal tract suggests the possibility that chronic gastro-enteritis in older dogs may be due to a prolonged mild deficiency of the blacktongue preventing factor.

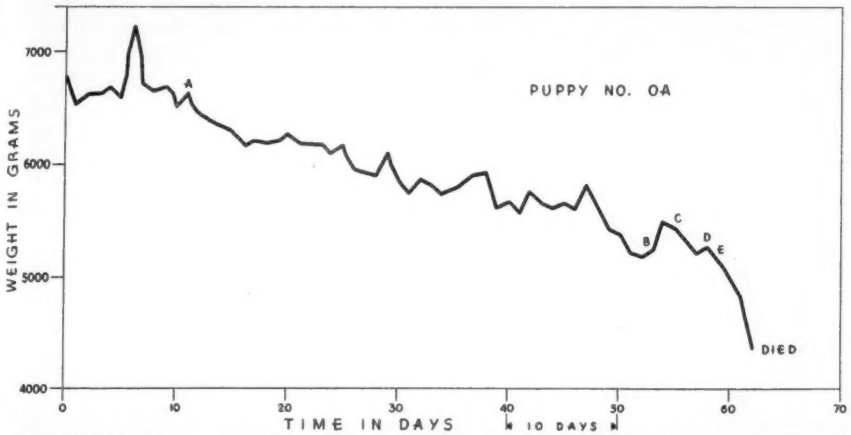


Fig. 41. This puppy received the Goldberger diet plus daily injections of 80 units of vitamin B_1, which were begun at "A". 10 grams of Flavex were given daily beginning at "B". At "C" 500 gamma lactoflavin were administered subcutaneously. At "D" marked diarrhea with mucus in feces was noted.

CLAIMS for certain dog foods are sometimes presented on the basis of the fact that several generations of rats have been raised successfully on this particular food. Inasmuch as the rat does not require the blacktongue preventing factor, it is obvious that such tests have no significance in demonstrating the nutritional value of any particular dog food. It is also quite obvious from these experiments that the blacktongue preventing factor is extremely important in the nutrition of the dog—in the nutrition of not only the mature dog, but in the young puppy.

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EDITOR'S NOTE.—This is the sixth of a series of eight articles on Vitamins and Distemper, by the same author. The seventh installment of this important discussion will appear in an early issue.

* * * *

A milk cow is prone to ailment when feed is poor throughout the winter, lacking proper nutritional elements. Spring opens up and pastures become good, increasing the flow of milk. Hypoglycemia is the result.—*W. B. Redman.*

Drugs Used In Veterinary Medicine

IV CALCIUM

By VICTOR LEWITUS, New York
Instructor in Materia Medica,
Columbia University College of Pharmacy,
Associate Professor in Materia Medica,
First Institute in Podiatry

Historical Sketch.—Calcium is one of the most versatile elements in medicine. Although the element itself has been isolated only in recent times, its compounds were long used in one way or another by ancient peoples. This occurred long before its identity had been established. That the metal should have had an early start along its career is not surprising, for the earth's crust is variously estimated to contain calcium to the extent of about 3.5%—in compound form of course.

The Roman architect Vitruvius, whom Pliny often quotes as an authority, described in great detail in his "Ten Books on Architecture" the early uses to which limestone was put. The preparation of lime from "burning stone", together with the principle of slaking was well understood in those times—and that was in the first century B.C.! Near Mount Vesuvius, a kind of "natural cement" was discovered, and according to Vitruvius "piers built of it in the sea, set hard under water." He further observed that "gypsum sets too rapidly and thus interferes with even drying" and this is the very plaster of Paris that we use today.

Although records of the medicinal use of calcium by the Romans are scanty, the employment of limestone or related products by the ancients of other regions appears to have been rather common. For example, the Egyptians are known to have had an extensive materia medica. Although we often turn up our noses, literally speaking, when we examine the list of items, such as lizards, snakes, various dungs and desiccated mummy, we must admit that there were included drugs which to this day have established their worth. Amongst these latter we find calcined hartshorn and bones, besides many vegetable drugs.

"In ancient Japanese and Chinese records, one finds likewise an application of calcium therapy which parallels in many respects modern usage. Everyone knows the story of the burnt sponge used to this day in China—for its iodine content as well as the calcareous materials that compose it. Ground sea-horses are also widely employed.

The mineral drugs of these Oriental peoples, point to the superstition of Taoism and the magic of the alchemists. One of the prescriptions found in the "Pen Ts'ao" (according to Dr. William R. Morse) reads like this: "If a man is restless and hysterical, when he wishes to sleep, and it is requisite to put him to rest, let the ashes of a skull be mingled with water and given him.....and he will be cured." Does not this have some bearing on modern concepts of calcium therapy? Even powdered Stalactites (a form of calcium carbonate from caverns) were used by them as a general tonic stimulant; and deer horns ("in the velvet") were also employed. Powdered bones were given for simple and compound fractures as well as for diseased bones and ulcers which points to their early scientific attack on deficiency disease.

Although calcium, in the form of chalk, was recognized as early as 1650 in the London Pharmacopeia and in the first edition (1820) of the United States Pharmacopeia, conscientious work on its place in plant and animal life did not begin until around 1856 when Salm-Horstmar proved the element was essential to seed-bearing plants. To Sydney Ringer, however, goes much of the credit for early discoveries of calcium requirements in the animal, though Morunowicz (in 1875) first observed that an aqueous extract of ash would support the heart when used as a perfusate. It was in 1883 that Ringer, while studying the effect of various salts on the life of aquatic organisms, observed that when fish were placed in distilled water their calcium

content and other salts were extracted from them, and in addition the mucous cells and epithelium of the gills became detached. He tried similar experiments with fresh-water Tubifex worms, finding that without calcium they would easily disintegrate whereas when the deficiency was supplied, the animals thrived as though they were in their natural habitat.

Herbst, in 1900, confirmed Ringer's findings, using sea-urchin larvae and allowing dissociation of the epithelial cells to occur up to a certain point, and then, by adding calcium salts, causing the cells to adhere together more intimately.

Of greater import, were Ringer's later findings with cardio-therapeutic effects of calcium. Using solutions of sodium, calcium and potassium salts he proved that if the calcium concentration became very low, contractions would lessen and the heart would finally stop in diastole. On the other hand, increasing the calcium content would augment contraction and if excess were added the heart would stop in a state of tonic contraction, the familiar systolic "calcium rigor."

Howell later found that the heart stored calcium in its muscular walls; and Arima noticed that in administering a calcium-free perfusion, the heart would cease contracting for a time and then spontaneously resume again since calcium was then found in the originally calcium-free fluid. Boehm in 1913, and Lloyd in 1929 found that calcium salts caused an increased strength of systole, a more regular rhythm, and better coordination between auricle and ventricle.

Until the present generation when nutritional studies on man and other animals came to the forefront, the place of minerals in the economy of the organism was not well understood. There is no doubt in the writer's mind that the discovery of the vitamins had much to do with assigning to calcium its present rôle. One need only study the work of such authorities as Dryerre and Grieg to conclude how necessary it is to correlate our findings to other fields in modern research before making conclusions, for careful consideration of the endocrine functions brought their results with calcium therapy of milk fever to a head

Calcium therapy in veterinary medicine is today one of the shining stars of our times. It will probably increase its hold within this field as time goes on, for there is much still to be learned regarding its greater assimilation, as with iron therapy in human medicine.

General Action of Calcium

Calcium compounds, according to the most recent consensus of opinion amongst medical men and veterinarians, serve to perform the important functions of forming the structure of bones, regulating nervous and muscular activity, and aiding in the coagulation of blood when in the proper concentrations. In certain diseases such as osteoporosis, osteomalacia and rickets the lack of proper deposition of calcium in the bones is, contrary to former belief, not considered due to a shortage of calcium itself but to other factors such as lack of vitamins D or parathyroid hypo-functioning; so that in these instances calcium therapy is next to worthless. It is only in rare cases that insufficient supply of calcium is a concommitant cause. When the calcium content of the blood is low, as in parathyroid tetany, the administration of calcium salts results in a temporary increase in blood calcium and a cessation of symptoms, but unless the cause is removed the calcium level sinks hopelessly. The administration of the parathyroid hormone has been found to increase blood calcium even without an added supply of calcium. This indicates that an animal that has a normal diet and proper endocrine function may utilize at least in part the calcium deposited in the skeleton.

Calcium lessens certain transudation processes in the animal body. For this reason some practioners have used it to treat certain types of edema, but its value here may be questioned.

Intravenous administration of calcium has been shown to cut down peristaltic movements and for this reason there is some promise for its application in case of

painful conditions of the biliary passages, the ureters and the intestinal tract.

Calcium has been reported as being effective in certain dermatoses such as erythemas and lichen, but more research must be done along these lines.

Deficiency of calcium in the circulation ends in neuro-muscular excitability (as in tetany) and this irritability is promptly decreased when the deficiency is removed.

Intravenous infusions of soluble calcium salts cause a constriction of blood vessels and usually a myotic effect on the pupil.

Calcium is necessary for proper blood coagulation, but an excess increases the coagulation time. This is indicated by the fact, that in cases of purpura, intestinal hemorrhage, and other hemorrhagic conditions the blood calcium is, as a rule, normal. When salts such as calcium chloride are administered, the basic portion is excreted mostly through the digestive tract, whereas the acid portion decreases the alkali reserve and increases the chances for acidosis, especially with large doses. The acidity of the urine is always increased by calcium, especially the chloride.

Some recent work has shown that the serum calcium consists of a diffusible and non-diffusible fraction. About half is diffusible and only a small portion of that is ionizable. The serum calcium is therefore not a true index of calcium levels since it is probable that the ionic portion is the active fraction.

Absorption of calcium, when given orally, takes place in the intestine. This is diminished by excess phosphorus in food which forms the unavailable tribasic calcium phosphate (hence the need for proper calcium-phosphorus ratio). An excess of fat in the diet forms insoluble calcium soaps. Intestinal alkalinity prevents absorption.

Zucker, *et al.* produced rickets in rats by adding sodium bicarbonate to the diet. Diarrhea depletes the system of calcium through discharge of the element, as does also excessive roughage. Intestinal acidity or added lactose because of lactic acid formation favors the absorption of calcium. The most readily available form of calcium appears to be in milk—pasteurization changes the calcium salts somewhat to insoluble forms.

The use of calcium salts when carbon tetrachloride is used tends to lessen the toxicity of the latter. The calcium salts are antidotal to oxalic acid and the soluble oxalates. Heller and Haddad have indicated the possibilty of depletion of calcium when magnesium salts were persistently administered. This tended to cause a sedative effect upon the nervous system of epileptics, when magnesium sulphate was used. High intake of magnesium, furthermore, has been found to lessen calcium storage.

Intravenous doses, when in excess, may prove fatal by paralysis of the heart and central nervous system. It has been shown recently that it is very hazardous to administer calcium salts intravenously after Digitalis has been given. This is probably true for other cardiac tonics or stimulants also. Rapid intravenous injections are also very dangerous with this group of drugs. The *slow* intravenous injection of calcium is strongly recommended.

Calcium Compounds Used Externally

Calcium Sulphate, Exsiccated.—Dried Gypsum. —$CaSO_4$.—This is the familiar Plaster of Paris. It is used only as a preparation for fixation bandages. (Some use it for chicken louse infestation).

Lime. (Quick Lime).—CaO.—This is used in the preparation of "slaked lime" by the addition of water; thus by dilution forming "lime water." Used alone as a disinfectant in stables and outhouses. Used in preparing Solution of Sulfurated Lime, (N. F.) for depilatory and dermatological conditions.

Lime and Sulphur Dip.—This is very effective against scabies or mange of horses and sheep: but not against lice or ticks. Directions for its preparation and use may be found in U. S. D. A. Farmer's Bulletin No. 713.

For dipping sheep, lime and sulphur dip is made in the proportion of eight pounds unslaked lime and 24 pounds of flowers of sulphur to 100 gallons of water. Place the lime in a shallow watertight box, and add enough water to form a thin paste. Sift the sulphur into it and mix until a paste (like mortar) is formed, adding water as required. Add this paste to 30 gallons of boiling water and boil for two hours, adding water to

keep up the original volume. Boiling and stirring until all sulphur disappears from the surface is essential. After the two-hour period of boiling, the liquid should have a dark-amber or chocolate color. After that it should be transferred cautiously to a water-tight barrel, which has an outlet (stoppered) four inches from the bottom. When the liquid fully settles, the sediment is at the bottom and the clear liquid on top. (The sediment must not be stirred in since it will injure the wool and the eyes of the sheep). When fully settled, draw off into the dipping vat and add enough warm water to make 100 gallons. When mixed and cooked as specified, the concentrate is three and one-third times the required strength for the dip in the vat, so that to every 30 gallons of such concentrate, 70 gallons of warm water should be added to make a dip of required strength.

For dipping horses or for application to these animals with a brush, make the dip according to the directions for sheep, except use 12 pounds of unslaked lime and 24 pounds of sulphur to 100 gallons of water (U.S.D.A. Circ. 148).

Calcium Hydroxide (Slaked Lime).—$Ca(OH)_2$.—A soft, white, crystalline powder, with an alkaline, slightly bitter taste. Slightly soluble in water (1:630), but insoluble in alcohol.

Slaked lime is used in making Solution of Calcium Hydroxide (N. F.) or lime water (0.15% $Ca(OH)_2$). It is used internally as an antacid and externally as a local sedative.

Lime Liniment (N. F.).—This is the well known carron oil—a 50% mixture of lime water and linseed oil—a soothing lotion, formerly used in the treatment of burns, but requires antisepsis.

Calamine Liniment (N. F.).—An emulsion of calamine, zinc oxide, olive oil and lime water (an improvement over carron oil).

Calamine Lotion (N. F.).—Similar to last-mentioned except that the olive oil is replaced with glycerine. *Phenolated Calamine Lotion* (N. F.) has 1% phenol added.

All of the foregoing preparations of slaked lime are used for their local soothing action on pruritic and erythematous conditions of the skin such as mange, or scabies, eczema, ear canker, etc.

Calcium Compounds Used Internally

For recent discussions of minerals, especially calcium, phosphorus and vitamin D relationships, refer to:

"Meeting the Mineral Needs of Farm Animals," L. A. Maynard, Cornell Extension Bulletin No. 350, April 1936.

"Feeding Chickens," U. S. D. A. Farmer's Bulletin No. 1541, May 1932.

"Nutrition in Relation to Animal Health," R. M. Bethke, *Veterinary Medicine*, 30:7, July, 1935.

"Conditions Mentioned in the Horse Breeding Acts," W. M. Mitchell, *Veterinary Medicine*, 31:11, November, 1936.

Prepared Chalk (Creta Praeparata) (Drop Chalk).—Chiefly $CaCO_3$.—This preparation is used mainly as an antacid by oral administration of the following preparations (also recommended for diarrhea): Chalk Mixture (U.S.P.); Compound Chalk Powder, (U.S.P.); Aromatic Chalk Powder, (N.F.).

Precipitated Chalk (Precipitated Calcium Carbonate).—$CaCO_3$.—This is practically identical to the above in composition and uses (in human medicine additionally used as a dentifrice powder).

Precipitated Calcium Phosphate. — $Ca_3(PO_4)_2$. —Tribasic.—This was formerly used like the calcium carbonate, but it is more insoluble than the latter. (The dibasic form, $CaHPO_4$, is said to be more available).

Tribasic calcium phosphate has certain advantages over alkaline hydroxides such as magnesium hydroxide or sodium bicarbonate since, because of its insolubility, it tends to neutralize excess acidity in the stomach but it produces less systemic alkalization. It causes constipation. Some of the calcium is absorbed and the drug may be used with expectation of some therapeutic effect systemically.

Calcium Chloride, U. S. P.—$CaCl_2 \cdot 2H_2O$.—This is a hydrated salt containing about 80% $CaCl_2$. It occurs as white translucent fragments, odorless, with a sharp saline taste. It is very deliquescent and must be kept in stoppered bottles. It is freely soluble in water and also in alcohol. It is incompatible with soluble carbonates, phosphates and sulphates producing insoluble calcium salts. The uses are similar to those described under calcium gluconate, but the drug is much more irritant to the animal and for this reason is not so popular. Its taste is disagreeable and the urine becomes acidified with it.

"Afenil" or Calcium Chloride Urea. — $CaCl_2.4(NH_2)_2CO$.—Is a new remedy having actions similar to calcium chloride. It is claimed that intravenous solutions are less irritant and better tolerated.

Calcium Lactate U. S. P. — $Ca(CH_3.CHOH.COO)_2.5H_2O$.—This is a white, crystalline powder or mass, odorless and nearly tasteless; soluble in water (1:20) and almost insoluble in alcohol. It is incompatible with carbonates and sulphates, forming insoluble salts. It is less irritant than the calcium chloride and therefore better adapted to hypodermic administration. Its action resembles closely that described under calcium gluconate.

Calcium Lactophosphate N. F. — This is prepared as an elixir and syrup. It is similar but with no advantage over the calcium lactate and is not adapted to hypodermic use.

Calcium Gluconate U. S. P.—$C_{12}H_{22}O_{14}Ca.H_2O$. —The normal calcium salt of gluconic acid, yielding about 12.6% CaO. It consists of white, odorless, tasteless, granules or powder, slowly soluble

in cold water (1:30) and more so in hot water (1:4 or 1:5) from which it will crystallize on cooling. It is insoluble in alcohol and fat solvents.

Calcium gluconate solutions have been rendered more stable by combination with boric acid forming calcium borogluconate. The method described by Hayden of the Cornell clinic, consists of mixing four grams of boric acid with each 100cc of distilled water which when used with the gluconate may be made up to 40% strength. The gluconate is added to the boiling boric acid solution, allowed to boil slowly for a few minutes and immediately strained through filter papers into sterile, good quality glass bottles. According to Thorshaug, a few drops of alcoholic thymol solution added to the above will add to its keeping qualities. Some recommend the addition of 0.5% phenol as the preservative. Sixty grams of calcium gluconate, 12gm of boric acid and distilled water q.s. 300cc should be used.

The indications for this most remarkable drug, according to Merillat, are as follows:

I. *Nervous Syndromes.*—Convulsions, lactation eclampsia, running fits, paraplegia or posterior paralysis, milk fever of cows (parturient paresis), pregnancy disease of ewes, systemic depressions, etc.

II. *Toxemias.*—Acetonemia of the dairy cow, azoturia of the horse, diabetes of the dog, etc.

III. *Calcium Deficiency.*—Rickets, osteomalacia, fragility of the skeleton, etc.

This drug is restricted in its application to non-infectious nervous syndromes, and therefore is not indicated in cases of rabies, "mad itch," (pseudo rabies, Aujeszky's disease), forage poisoning, canine distemper and leg paralysis of fowl, since these have their origin in some virus or other infective agent.

This same authority states that this drug is to be injected intravenously or intraperitoneally in dogs and cats, instead of transcutaneously. He mentions that "the dose is too bulky for intramuscular delivery, and in subcutaneous injections, the solution should be distributed over a considerable area and absorption activated by massage. Intravenous injections delivered slowly remove much of the risk of provoking cardiac crisis."

Calcium Levulinate (Calevate — Crookes Laboratories) (Calcium Levu-Gluconate — National Drug Company).—Because of the disadvantage of limited solubility and possible crystallization of ordinary calcium gluconate and because of the low concentration of calcium and the bulkiness of the solutions, a new product, Calcium Levulinate $(CH_3CO.CH_2CH_2COO)_2Ca.2H_2O$, has been introduced recently. It is very stable in water; unaffected at 100° C. or when chilled to freezing temperatures. It contains almost 15% calcium on an anhydrous basis, the gluconate containing but 9.3%. It is claimed to be less toxic also. The solutions, however, are claimed to be stable up to 30%. Calevate (Crookes) contains 13.1% calcium combined with hydrolized gluconic acid.

Calcium Malate.—According to Podolsky, the gluconate is the best of the calcium salts but due to poor stability of solutions, its bulky dose and the fact that the difference between the effective and toxic doses is not very great, there is added danger. He claims that the malate is the most suitable agent, for this drug in fine colloidal dispersion, affords slower and more effective release of the calcium ion. The slower release, he claims, insures not only greater retention but also prevents the reactions due to the shock of too rapid release of the therapeutic agent. It is stable in respect to temperature changes and ideal for intravenous medication.

Calcium Glycerophosphate (N. F.) — $CaC_3H_5(OH)_2PO_4$.—This, together with its preparation the elixir of calcium and sodium glycerophosphates was at one time esteemed as a tonic for the nervous system, but there is little evidence that it is so selective in action.

Calcium Hypophosphite (N. F.) — $Ca(PH_2O_2)_2$.—This, together with the syrup and the compound syrup, was at one time considered of special value in tuberculosis and as a reconstructive tonic, but there is no reliable clinical evidence in this regard. It may be considered as a vehicle for oral calcium medication.

Other official salts of calcium such as calcium peroxide, calcium bromide, calcium creosotate and calcium iodobehenate are not used especially for their calcium ions but for other effects.

"Broken Back" in Dog

An x-ray photograph of an airedale terrier which had been knocked down by a car disclosed a serious dislocation of the seventh dorsal vertebra.[88] The supraspinous process of the eighth vertebra appeared to be fractured. The clinical symptoms were posterior paralysis, great distress, especially when being moved. The near fore-limb was extended in a stiff manner.

[88] Kirk, H. 1937. Spinal dislocation. *Vet. Rec.* 49:235-236.

A Study of the Relative Merits of Pellets and Mash Poultry Feeds*

By J. W. PATTON, H. H. BUSKIRK and L. A. RAULS,
East Lansing, Michigan

Introduction

The increasing popularity of poultry feeds in pellet form made it appear pertinent to initiate a study inquiring into the relative merits of the same feed formula varying only in physical form (mash and pellet) under controlled conditions.

Object

The object of this experiment was to find out the relative nutritional value of the same feed, different only in physical form, as determined chiefly by the growth of single comb white leghorn chickens from the date of hatching to the end of the ninth week for the cockerels and the end of the twelfth week for the pullets and under controlled conditions.

Method

Briefly, the method was to divide one hundred and twenty-one (121) day-old single comb white leghorn chicks into two, approximately equal, groups; to standardize brooding and general conditions of management, and to keep accurate data on feed and water consumption and on growth by periodic weighing.

Conditions of the Experiment

1. Chicks.—The chicks were hatched from eggs laid by disease-free hens from a basic breeding flock in the possession of this laboratory.† The hens, like these chicks, were on a definite bio-assayed diet. The hens were trapnested and the eggs were pedigree-hatched during incubation. The resulting chicks from each hen were divided so that each hen was equally represented on both the mash and pellet experiments. This precaution was taken to equalize, as much as possible, any difference that might result from genetic factors not under our control.

The chicks were wing-banded and appropriate records were kept of their parentage. They were weighed when 24 hours old and at this time they were divided into two groups, 1A (pellets) and 2A (mash).

2. Brooding.—Each lot of chicks was placed under electrically heated and controlled hovers with a capacity of 250, day-old chicks. These hovers were placed side by side in two specially constructed 5 x 8 feet, wire-screened pens in a basement room of the laboratory. This room was chosen because temperature and humidity could be controlled there. The brooding room provided semi-darkness so that the length of the chick day could be regulated. This, together with the absence of other extraneous factors (e.g. sunlight, etc.), enabled us to control the experiment in certain essential respects. The initial brooding temperature was 95° F. and this was reduced 5° weekly for five weeks, at which time the brooders were removed. The room temperature varied between 70° and 75° F. throughout the experiment, whereas the relative humidity varied between 50 and 60.

3. Feed.—The feeding units consisted of two pens. Pen 1A was placed on pellets and Pen 2A on mash. Each pen received this feed exclusviely. The feed used in these tests was furnished by Nutrena Mills, Inc., of Kansas City, Kansas, and was of regular manufacture. The feeds were bio-assayed by this laboratory for vitamin activity per pound as follows:

Vitamin A....6324 International Units.
Vitamin B.... 300 Chase-Sherman Units.*
Vitamin D....175 International Units.*
Vitamin G (B_2) 300 Bourquin-Sherman Units.*

*Assays were run at the levels given. Results, however, showed that the feeds carried unitage in excess of these amounts.

The chemical analysis of the feed was as follows (from feed tag):

Protein	not less than	18.0%
Fat	not less than	3.5%
Fiber	not more than	5.0%
Carbohydrates		
Nitrogen-free extract	not less than	51.0%

*Made under a Grant from Nutrena Mills, Inc., Kansas City, Kansas.
†Patton Biological Laboratories, East Lansing, Mich.

TABLE NO. I. GROWTH OF WHITE LEGHORN MALES ON CHICK MASH PELLETS

Hen No.	Chick Number	Initial Weight	Weeks 1	2	3	4	5	6	7	8	9
367	692	35.5	48.5	79.0	125.0	198.0	273.0	380.0	514.0	624.0	740.0
"	693	37.5	59.0	109.0	171.0	267.5	335.0	457.0	564.0	679.0	804.0
"	694	35.0	57.0	102.5	168.0	263.0	334.0	440.0	548.0	634.0	766.0
"	696	34.0	47.0	73.0	111.0	162.0	222.0	304.0	409.0	476.0	596.0
370	702	39.0	69.5	128.0	207.0	312.0	377.0	519.5	603.0	655.0	832.0
360	712	37.0	63.0	100.0	159.5	244.0	310.0	390.0	523.0	620.0	713.0
"	713	40.5	69.0	115.0	187.0	296.0	382.0	487.0	597.0	670.0	810.0
352	738	36.5	62.5	116.5	175.0	259.0	320.0	428.0	550.0	624.0	752.0
"	739	34.5	55.0	101.0	163.0	244.0	305.0	400.0	533.0	633.0	681.0
353	740	41.0	46.5	77.0	103.5	135.0	160.0	200.0	247.0	300.0	365.0
354	751	36.5	68.0	124.5	197.5	290.0	370.0	473.0	587.0	687.0	810.0
388	752	37.5	54.0	104.0	181.5	304.5	381.0	515.0	709.0	807.0	1004.0
"	753	41.0	66.0	120.0	201.0	312.5	372.0	501.0	664.0	785.0	914.0
369	766	44.5	52.0	80.0	118.0	164.0	220.0	290.5	365.0	414.0	502.0
104	769	45.5	73.0	129.5	209.5	313.0	380.0	505.0	618.0	718.0	861.0
376	777	39.5	64.0	103.0	164.0	245.0	302.0	392.0	526.0	601.0	702.0
"	780	37.0	66.5	127.5	195.0	296.0	360.0	452.5	594.0	713.0	831.0
359	795	37.0	66.5	128.5	218.0	353.5	434.0	562.5	686.0	736.0	885.0
"	796	36.0	74.0	143.0	223.0	344.0	403.0	510.0	612.0	687.0	820.0
332	805	41.0	61.0	99.0	156.5	245.0	316.0	420.0	535.0	579.0	741.0
"	806	40.0	59.0	105.0	170.0	258.5	336.0	450.0	566.0	619.0	770.0
345	809	38.0	46.0	71.0	108.0	177.0	239.0	322.0	439.0	503.0	646.0
Average:		38.4	60.3	106.2	168.7	258.7	324.1	427.2	544.9	625.6	752.0

TABLE NO. II. GROWTH OF WHITE LEGHORN FEMALES ON CHICK MASH PELLETS

Hen No.	Chick Number	Initial Weight	Week 1	2	3	4	5	6	7	8	9	10	11	12
367	695	36.0	53.0	93.5	153.0	225.0	282.0	367.0	438.0	493.0	547.0	645.0	719.0	763.0
370	703	38.0	58.0	95.5	136.0	207.0	260.0	349.0	435.0	508.0	574.0	666.0	764.0	839.0
"	704	37.0	56.0	99.0	152.0	225.5	279.0	361.5	455.0	511.0	609.0	680.0	735.0	815.0
"	705	38.0	61.0	102.0	156.5	227.0	287.0	380.0	467.0	512.0	620.0	634.0	705.0	740.0
360	711	35.5	54.0	86.0	129.5	200.0	265.0	352.0	428.0	451.0	560.0	650.0	678.0	778.0
307	719	32.5	63.0	101.0	161.0	254.0	320.0	417.5	505.0	515.0	611.0	685.0	740.0	831.0
"	720	36.0	54.0	88.0	144.5	223.5	267.0	370.0	471.0	386.0	Died			
"	721	38.5	66.0	119.5	170.0	236.0	300.0	380.0	460.0	526.0	596.0	669.0	733.0	830.0
412	726	40.5	72.5	130.0	206.5	317.0	392.0	500.0	609.0	644.0	765.0	846.0	925.0	1020.0
"	727	33.0	47.0	82.0	133.0	203.0	261.0	352.0	448.0	501.0	618.0	697.0	744.0	825.0
347	730	36.0	56.0	100.0	150.0	220.0	275.0	345.0	430.0	488.0	576.0	624.0	686.0	750.0
"	731	37.0	64.0	106.5	159.5	235.0	305.0	368.0	475.0	551.0	628.0	700.0	738.0	803.0
353	741	37.0	51.0	78.5	111.0	153.0	Died							
354	748	37.0	58.5	103.0	166.0	242.0	310.0	387.0	450.0	485.0	567.0	650.0	698.0	752.0
"	749	39.5	53.0	95.0	146.5	210.0	254.0	336.5	421.0	470.0	547.0	583.0	644.0	731.0
"	750	34.5	51.0	94.0	150.0	225.0	275.0	337.5	425.0	493.0	557.0	622.0	690.0	764.0
314	758	40.0	57.0	101.0	149.0	223.0	292.0	360.0	467.0	500.0	603.0	670.0	745.0	850.0
369	767	49.0	49.0	85.0	141.0	271.0	275.0	354.5	451.0	543.0	633.0	703.0	776.0	847.0
184	768	42.5	66.0	128.0	200.0	292.0	381.0	483.5	610.0	648.0	728.0	761.0	846.0	879.0
345	774	33.5	62.0	117.5	178.0	257.0	326.0	424.0	526.0	604.0	737.0	845.0	939.0	1013.0
"	775	37.5	44.0	67.5	93.0	132.0	181.0	237.0	322.0	390.0	432.0	510.0	536.0	600.0
376	778	38.5	61.5	103.5	160.0	234.0	279.0	355.0	455.0	531.0	605.0	664.0	690.0	797.0
"	779	37.5	52.0	89.5	146.5	235.0	285.0	320.5	400.0	486.0	546.0	610.0	661.0	744.0
"	781	39.0	55.0	96.0	140.5	189.0	230.0	298.5	372.0	423.0	482.0	526.0	586.0	647.0
359	793	43.0	64.5	113.0	171.0	265.0	317.0	430.0	552.0	637.0	760.0	857.0	910.0	992.0
"	794	38.0	63.5	124.0	198.0	295.5	352.0	448.0	539.0	588.0	683.0	723.0	740.0	820.0
"	797	37.5	70.0	133.0	205.0	304.0	368.0	452.5	594.0	636.0	749.0	881.0	936.0	996.0
332	804	39.5	36.0	60.0	107.0	154.0	204.0	264.0	373.0	450.0	515.0	579.0	672.0	740.0
269	826	38.0	46.0	71.0	108.0	160.0	212.0	276.0	360.0	402.0	498.0	562.0	618.0	679.0
Average:		37.9	56.7	98.7	152.4	228.1	286.9	368.1	462.1	513.3	605.4	675.6	793.0	809.0

The pellets and mash came to us at date of manufacture and at intervals which enabled us to use both with assurance that there was no appreciable difference on account of age. The age of the feed as fed varied between ten days and three weeks. Thus uniformity was maintained to overcome whatever influence if any the age factor might exert.

4. Feeding.—The feed was placed in metal feeders designed to prevent spilling or waste. A sufficient number of feeders were used to provide adequate space for all chicks at all times. A weighed quantity of feed was placed in the feeders each morning. This amount varied with the age and requirements of the chicks. In each case, however, the amount was so gauged as to provide adequate amounts of feed at all times. Weekly weighings and tabulations were made. Because precautions were taken to prevent spilling or waste of either pellets or mash, such loss was negligible. However, whatever loss did occur was re-

TABLE NO. III. GROWTH OF WHITE LEGHORN MALES ON CHICK MASH

Hen No.	Chick Number	Initial Weight	Weeks 1	2	3	4	5	6	7	8	9
367	697	39.0	60.0	91.0	158.0	245.0	323.0	422.5	517.0	660.0	782.0
370	706	39.0	58.0	91.0	150.0	230.0	308.0	400.0	516.0	649.0	780.0
"	707	40.0	64.0	111.0	177.0	260.0	344.0	406.0	482.0	573.0	686.0
360	710	36.0	64.5	111.0	173.5	243.0	314.0	384.0	467.0	571.0	698.0
"	714	35.5	65.0	117.5	168.5	244.0	301.0	400.0	473.0	551.0	645.0
"	715	40.0	63.5	109.5	163.0	241.0	315.0	406.0	497.0	551.0	714.0
347	732	37.0	65.0	116.0	181.5	268.0	349.0	446.0	544.0	652.0	737.0
"	734	36.0	61.5	107.5	161.0	243.0	330.0	420.0	516.0	610.0	679.0
353	742	37.5	51.0	79.0	121.0	170.0	216.0	279.5	345.0	409.0	540.0
354	744	38.0	57.5	101.0	157.5	232.0	280.0	370.0	445.0	547.0	599.0
"	745	36.5	55.0	109.0	181.0	270.0	350.0	460.0	538.0	640.0	761.0
"	747	39.5	61.5	90.5	144.5	212.0	278.0	347.5	447.0	545.0	663.0
388	754	39.0	61.0	110.0	174.5	243.0	319.0	428.5	505.0	640.0	704.0
314	755	47.0	62.0	108.0	161.0	219.0	307.0	392.5	485.0	545.0	644.0
"	756	44.0	67.0	111.0	167.0	236.0	319.0	422.0	466.0	576.0	612.0
369	763	44.5	61.0	111.0	173.5	248.0	324.0	410.0	477.0	565.0	668.0
345	771	35.5	60.0	104.5	164.5	242.0	324.0	420.0	522.0	594.0	714.0
"	773	40.0	52.5	86.5	124.0	192.0	262.0	323.5	365.0	496.0	593.0
376	785	35.0	57.5	113.0	182.0	281.0	361.0	452.5	524.0	625.0	719.0
"	786	41.5	62.0	102.5	157.0	232.0	300.0	385.5	485.0	586.0	664.0
359	788	38.0	69.0	130.0	211.5	307.0	388.0	498.0	551.0	691.0	740.0
"	789	38.0	65.0	121.5	178.5	245.0	311.0	390.0	462.0	593.0	695.0
"	791	39.5	78.0	149.0	229.0	341.0	449.0	570.0	646.0	795.0	916.0
322	801	39.5	64.0	100.0	162.0	233.0	291.0	384.5	426.0	493.0	623.0
"	803	43.0	64.0	107.0	160.5	247.0	333.0	425.0	525.0	603.0	749.0
104	807	38.0	56.0	101.0	174.0	265.0	353.0	459.0	563.0	670.0	799.0
269	810	38.0	58.5	100.0	151.0	208.0	246.0	337.0	424.0	550.0	665.0
Average:		39.1	61.3	107.0	166.9	244.3	318.3	408.9	489.4	591.9	695.9

TABLE NO. IV. GROWTH OF WHITE LEGHORN FEMALES ON CHICK MASH

Hen No.	Chick Number	Initial Weight	Week 1	2	3	4	5	6	7	8	9	10	11	12
367	698	38.5	63.0	108.5	169.0	240.0	322.0	404.0	459.0	542.0	619.0	689.0	709.0	797.0
"	699	35.5	62.0	106.5	160.0	231.0	307.0	384.0	445.0	482.0	566.0	652.0	692.0	780.0
"	700	35.5	57.0	90.0	129.0	188.0	250.0	320.0	390.0	455.0	540.0	612.0	680.0	700.0
370	708	39.0	57.0	95.5	141.5	203.0	240.0	336.0	401.0	470.0	566.0	621.0	657.0	683.0
360	709	38.0	65.5	118.0	185.5	250.0	344.0	430.0	479.0	572.0	648.0	700.0	722.0	792.0
307	716	39.0	65.0	117.5	175.5	254.0	320.0	391.0	428.0	501.0	590.0	650.0	700.0	752.0
"	717	35.0	49.5	74.0	112.0	172.0	225.0	382.0	343.0	395.0	482.0	566.0	612.0	684.0
"	718	36.0	50.0	73.0	114.0	165.0	201.0	278.5	328.0	393.0	351.0	480.0	509.0	578.0
412	724	40.0	67.0	115.0	186.0	257.0	336.0	395.0	443.0	523.0	620.0	671.0	724.0	782.0
"	725	38.5	59.0	88.0	131.0	204.0	260.0	360.0	403.0	502.0	600.0	670.0	722.0	795.0
347	733	36.5	61.0	114.0	179.0	253.0	318.0	392.0	415.0	484.0	548.0	596.0	621.0	643.0
"	735	38.0	67.0	116.0	175.0	255.0	336.0	390.0	463.0	563.0	636.0	678.0	646.0	683.0
361	736	38.0	56.0	97.0	139.5	193.0	253.0	310.0	370.0	450.0	515.0	465.0	590.0	641.0
352	737	34.0	60.5	104.0	155.5	234.0	292.0	375.5	426.0	418.0	534.0	606.0	652.0	708.0
353	743	38.5	51.5	70.0	85.0	107.0	124.0	150.0	171.0	192.0	Died			
"	811	38.0	42.0	69.0	104.0	150.0	199.0	259.0	310.0	376.0	455.0	509.0	558.0	593.0
354	746	33.5	50.0	98.0	159.0	242.0	327.0	425.0	489.0	584.0	675.0	725.0	750.0	840.0
314	757	42.0	61.0	106.0	158.0	226.0	297.0	372.0	426.0	497.0	584.0	650.0	689.0	707.0
269	762	45.5	53.5	96.0	147.0	230.0	282.0	344.5	413.0	487.0	589.0	656.0	748.0	760.0
369	764	45.0	61.0	102.0	158.5	233.0	299.0	366.5	453.0	542.0	589.0	608.0	682.0	704.0
104	770	41.5	75.0	138.0	212.5	297.0	400.0	495.5	554.0	678.0	807.0	879.0	900.0	981.0
345	772	43.0	58.5	101.0	158.0	237.0	313.0	400.0	448.0	525.0	582.0	658.0	682.0	725.0
376	782	38.0	63.0	112.0	178.5	263.0	327.0	415.5	507.0	602.0	690.0	755.0	804.0	846.0
"	783	35.0	53.0	97.0	156.5	236.0	303.0	400.0	437.0	531.0	579.0	649.0	701.0	737.0
"	784	37.5	58.5	98.5	158.0	230.0	295.0	405.5	496.0	521.0	559.0	636.0	674.0	728.0
359	790	40.0	65.5	128.0	197.5	282.0	369.0	443.5	502.0	537.0	633.0	709.0	777.0	792.0
"	792	37.0	57.0	102.0	160.5	242.0	303.0	401.0	469.0	545.0	619.0	719.0	744.0	772.0
332	800	40.0	62.0	104.5	158.0	220.0	267.0	305.5	411.0	475.0	535.0	596.0	660.0	700.0
"	802	41.0	60.5	110.0	168.0	237.0	320.0	407.0	468.0	432.0	603.0	681.0	675.0	798.0
Average:		38.5	59.4	101.7	155.6	225.2	290.6	335.8	426.7	492.2	582.6	649.4	686.4	739.3

corded and the necessary corrections on feed consumption were made.

5. Water.—Fresh tap water was provided each morning in a two-gallon fountain of a type designed to prevent spilling. This was checked twice daily and additional water added when necessary. The water consumption was recorded on a weight basis. The fountain and its contents were weighed at the time of filling and again immediately prior to refilling. No records on water consumption were kept until the commencement of the third week.

6. Lighting.—The influence of the length of the chick day on feed consumption and other controlling factors necessitated semi-darkness, so artificial illumination was provided to simulate daylight. Illumination was by a 60 watt ordinary Mazda electric light bulb in each pen. The lights were

TABLE V. FEED CONSUMPTION (GRAMS)

PELLETS

Average Initial Weight 38.2 gm.

Weeks	No. of Chicks	Total Feed	Average Feed Per Chick*	Average Weight of Chicks	Average Gain in Weight*	Ratio of Gain to Feed*
1	52	2510	48	58.5	20.3	1:2.3
2	52	5860	113	102.5	44.0	1:2.6
3	52	7880	152	160.6	58.1	1:2.6
4	51	11750	230	243.4	82.8	1:2.8
5	51	13600	267	305.5	62.1	1:4.3
6	51	16950	332	397.7	92.2	1:3.6
7	51	21300	418	503.5	105.8	1:4.0
8	51	17700	347	569.5	66.0	1:5.2
9	49	21250	434	678.7	109.2	1:4.0
			2341		640.5	1:3.7

MASH

Average Initial Weight 38.8 gm.

1	56	2750	49	60.4	21.6	1:2.3
2	56	6980	125	104.4	44.0	1:2.8
3	56	9770	175	161.3	56.9	1:3.1
4	56	12700	227	234.8	73.5	1:3.1
5	56	14850	265	304.5	69.7	1:3.8
6	56	18550	331	372.4	67.9	1:4.9
7	56	23450	419	458.1	85.7	1:4.9
8	56	23550	421	542.1	84.0	1:5.0
9	55	25200	459	639.3	97.2	1:4.7
			2471		600.5	1:4.1

* Weighted Averages.

TABLE VI. WATER CONSUMPTION (KG.)

PELLETS

Weeks	Number of Chicks	Total Water	Average Water Per Chick	Average Feed Per Chick*	Ratio of Feed to Water
		kg.	kg.	gm.	
3	52	14.5	.3	152	1:2.0
4	51	21.3	.4	230	1:1.7
5	51	24.2	.5	267	1:1.9
6	51	35.2	.7	332	1:2.1
7	51	38.4	.8	418	1:1.9
8	51	37.5	.7	347	1:2.0
9	49	45.9	.9	434	1:2.1
Totals			4.3	2180	1:2.0

MASH

		kg.	kg.	gm.	
3	56	13.3	.2	175	1:1.1
4	56	20.0	.4	227	1:1.3
5	56	28.1	.5	265	1:1.9
6	56	30.4	.5	331	1:1.5
7	56	32.1	.6	419	1:1.4
8	56	36.2	.6	421	1:1.4
9	55	41.0	.7	459	1:1.5
Totals			3.5	2297	1:1.5

* Taken from Table No. V.

turned on at 8 a.m. and shut off at 6 p.m.

7. Mortality.—Since the chicks were placed on this experiment when a day old and some death in day-old chicks is to be expected, all records of chicks which died between the initial weighing and the end of the first week were eliminated from this experiment. We wish to record, however, that out of the original 121 chicks placed on the experiment, 13 died between the first and fourth days. Records of these 13 chicks do not appear in this data.

Only three deaths occurred from the fourth day to the twelfth week of the experiment and these were pullets No. 741 and No. 720 on pellets and pullet No. 743 on mash. It is interesting to note that pullet No. 741 on pellets and pullet No. 743 on mash were from hen No. 353. In this and other experiments this hen has shown a decided genetic influence of lethal factors both during and following hatching. Note further that chicks Nos. 740 and 742, males, and chicks Nos. 741, 743 and 811, females, the progeny of this hen on this experiment, either died or failed to grow well. A further record of this hen and her progeny in another experiment, an experiment on hatchability and growth, will be given later.

8. Exercise.—The only exercise permitted the chicks was what they normally secured in their combination brooder and pen. The chicks, of course, were never permitted outside of their quarters.

9. Management in General.—Accurate feed consumption data is difficult to secure, even under carefully controlled conditions, so every effort was made to cut down the possibility of error. Therefore no grit, shell, charcoal or other supplements to a chick diet were allowed. The chicks were on a strict diet of weighed amounts of feed and water. The litter used was peat moss, chosen because it contrasted sharply with the feed and enabled us to check the litter carefully for waste feed. The cockerels were separated and disposed of at nine weeks. The pullets were carried on to the end of twelve weeks, at which time they were placed on range. Except as otherwise noted, commonly accepted principles and practices of poultry husbandry prevailed.

Discussion

It should be understood that this experiment was under strict laboratory control which included a division of the offspring (chicks) of individual hens between mash- and pellet-fed lots—split litters. The chicks were fed on a flock basis, and while this

TABLE NO. VII. SUMMARY

Type of Feed	Feed Consumed Per Chick*	Water Consumed Per Chick†	Average Growth of Flock**	Feed Per Unit of Gain
Pellet's	2341 gm.	4.3 kg.	678.7	3.7 gm.
Mash	2471 gm.	3.4 kg.	639.3	4.1 gm.

* Nine weeks.
† Seven weeks.
** Weighted average.

phase of the experiment closely simulates common poultry practices, it does not take into consideration the individual growth potentials beyond split litters.

As would be expected, a feed in pellet form is more easily eaten than a similar feed in mash form and this was our experience in these tests. The pellet-fed pen satisfied their appetite, or desire for feed, much more quickly than did the mash-fed pen. This, obviously, gave them a geater portion of their time for non-feeding activity.

It has been charged that much leisure is a factor in promoting such undesirable practices as cannabalism and feather-pulling. This has not been our experience in this, and other, closely-controlled research; notwithstanding the fact that conditions of management were ideal for the occurence of vices.

From our data it is calculated:

1. The mash-fed chicks consumed 5.55% more feed than pellet-fed chicks.

2. Weighted averages show the pellet-fed chicks gained 6.16% more than mash-fed chicks.

3. The mash-fed chicks consumed 10.81% more feed per unit of grain than the pellet-fed chicks.

4. The pellet-fed chicks consumed 22.86% more water than the mash-fed chicks.

Though this experiment indicates a superiority of the pellet-fed pen over the mash-fed pen along three lines of research namely, (1) growth, (2) gain in relation to feed consumption, (3) water consumption, it is less clearly defined why this difference exists. It will be noted that although the chicks on this experiment were maintained under strict laboratory conditions, growth on the whole for both pens was quite satisfactory and the mortality could be considered quite low.

Conclusions

It would appear from this experiment and data that a feed formula in pellet form offers nutritional advantages, as manifested by greater total growth and greater growth per unit of food consumed, over the same feed formula in mash form and with specific reference to single comb white leghorn chicks from one day to nine weeks of age.

It would appear further that the water requirement of pellet-fed chicks is greater than that of corresponding mash-fed chicks under controlled conditions and with specific reference to single comb white leghorn chicks from one day to nine weeks of age.

* * * *

Big Head in Sheep

So-called big head, thick ears, proteid anaphylaxis, or allergic reaction, prevailed from the middle of June to the middle of August, in Missouri and adjacent territory, during the past season.

This condition has been relatively common for the past several years. It is primarily a disease of lambs.

During the present outbreak the malady is more severe than it has been in recent years and the fatality rate has been higher.

As in former years, treatment was unsatisfactory. Local application of a soothing astringent ointment or oleaginous mixture on the head lesion is apparently of value. In addition to the local treatment, it is advisable to keep the affected and exposed sheep and lambs in a barn or shed in the shade and on dry feed.

Briefly, this ovine malady is an economic factor in the sheep industry and unless ways and means of control are solved it will probably be necessary to breed for winter lambing.

The major portion of the affected lambs manifested an icterus. This may be of significance in establishing the cause and developing a treatment.—*A. T. K.*

Abstracts

Periodic Ophthalmia an Allergy

When one examines[78] the parenchyma of the liver, spleen, kidneys, lungs, in a horse with periodic ophthalmia, one is apt to find lymphocytary cell aggregations around the vessels, especially in the peripheral parts of the liver, where they may occur in considerable numbers in the triangle of Glisson. Not infrequently the liver cells are degenerated. Similar foci of infiltration are located in the spleen, bringing about characteristic changes in the border cells and in the cells of the adventitia of the vessels. In the kidneys there are at times pronounced cell aggregations in the interstitial tissue and in the capillaries, and at times rather large foci. As a rule, the infiltrations are the least pronounced in the lung, but they may be found around the bronchioles and the bifurcation. On the whole the lesions affect the reticulo-endothelial system and the active mesenchyme of the vessels. The attack takes place through a single or a repeated inundation with toxic proteins, either in the form of bacteria or in the form of toxins. Immediate local infection with bacteria can be ruled out with certainty in periodic ophthalmia.

All observations point to a gradually occurring intoxication. The invasion with foreign protein is met by the reticulo-endothelial apparatus and the active mesenchyme, which cells use it and dissolve it; it is finally disposed of by the migratory cells. This takes place while the body is in a state of allergy. If now a new inundation, or a repeated inundation with foreign protein takes place, then the recticulo-endothelial apparatus is unable to dispose of it; the body is in a state of hyperergasia, and is highly sensitized against the foreign protein. If the inundations do not cease, then the vessels will be unable to dispose of the toxin, and an abnormal reaction, the so-called anaphylactic shock, takes place.

Numerous experiments and zoological observations indicate that the toxins which produce hyperergasia in horses originate from intestinal helminths, the ascarids and the strongylids, which usually are present together. The ascarids excrete a toxin which enters the circulatory system through the excoriations and ulcers caused by the strongylids. Possibly, the strongylids themselves may participate in the toxin production. For, Manninger (1930) has shown that the horse's own protein, when decomposed, possesses toxic properties and causes auto-intoxication. Such decomposed protein is produced by the strongylids in the process of digestion of the epithelium of the mucosa. The reticulo-endothelial system is continually put to the defense against toxin inundations, until finally the mechanism breaks down, and the injury becomes apparent, in the case under discussion as uveitis, etc.

Periodic ophthalmia occurs only in wet climates; it is unknown in arid regions. It is frequent in mines where the possibility of worm invasion is great. It is generally agreed that the disease is not hereditary, but there are indications that individuals exist which possess a constitutional disposition toward this disease.

Prophylactically the fight against periodic ophthalmia must be directed against the intestinal parasites: Vermifuge treatment; collection and sanitary disposal of the manure; clean water supply.

[78] Gmelin, W. 1936. Mondblindheit. (Periodic ophthalmia.) in Stang-Wirth, "Tierheilkunde und Tierzucht" vol. 11, pp. 446-450.

* * * *

Intracecal Injection for Whipworm

Having treated 21 cases of whipworm infestation by intracecal injection of santonin, Cagni[89] checked the outcome of the treatment by fecal examination over a certain period. He reports that 12 cases were possibly successful, three cases possibly suffered from reinfestation, and six cases were definite failures.

[89] Cangi, C. W. 1937. Treatment for whipworms in the dog by intracecal injection. *Cornell Vet.* 27:21-24.

Sexual Vices in Horses

Nymphomania in mares and masturbation in stallions are very disagreeable and annoying vices when encountered in the systematic training of race horses. Causes contributing to this hypersexuality are the heavy feeding of oats, the meticulous grooming, the boredom of being confined singly for hours, and the mingling of both sexes during training and races. Occosionally this hypersexuality occurs in such a violent form that the horse becomes unmanageable, and useless for work. So far, the remedial measures in vogue have either serious disadvantages, or are entirely inefficient.

An organ preparation, known as epiphysan (pineal extract), obtained from the pineal body of cattle has produced antilibidinous results in onanism of man. Hutschenreiter[80] employed this hormonal product in nine mares and seven stallions which had exhibited hypersexuality. The method of application was by subcutaneous injection. The dosage varied between 5cc and 10cc. The injection may be repeated within 24 hours.

Depending on the degree of sexuality, the sexual excitement is suppressed within one to three days; the suppression often lasts for many days. In addition to the anaphrodisiac action, the epiphysan exerted also a beneficial infiuence upon the general condition of the horse: the appetite and condition improved; the horse exhibited new vigor, and greater liveliness of temper.

All Milk Cows Harbor Streptococci

Studies of 46 udders[81] aseptically removed from cows and virgin heifers in which no evidence of mastitis infection could be detected by gross examination either prior to or subsequent to removal, revealed that all animals which had passed thru one or more lactation periods had mastitis streptococci in the mammary tissue. Fewer than half of the udders examined from virgin heifers contained these organisms.

Milk Alteration Due to Mastitis

No significant changes in chemical composition were found in samples of milk taken from cows with latent infections of mastitis so long as the milk remained normal in its physical appearance.[82] Slight differences include a decrease in lactose, specific gravity, skimmilk solids, and curd tension, while the chlorides and albumins were slightly increased, but these differences were no greater than variations in chemical composition of milk between two herds of the same breed. It is concluded that if all abnormal milk from inflamed, congested, or injured quarters is excluded, mixed milk is essentially normal in chemical composition.

Long Distance Breeding

Artificial insemination is one of the possibilities for economic improvement of livestock. Artificial insemination has been extensively employed in Russia, particularly in the breeding of cattle and sheep. The following report indicates that American cattle breeders are also interested in artificial breeding methods:

September 19th, 1936, seminal fluid was obtained from a Hereford bull in the United States and transported 8,000 miles by airplane, railroad and automobile to "La Merced" in the province of Buenos Aires, where it arrived safely and was used successfully September 25th, 1936, on seven cows.

According to Kingman, of Wyoming, from 2 to 4cc of semen can be recovered from a bull by massage of the accessory genital organs per rectum. He further states that "the proper temperature to maintain viability of sperm has been shown to be approximately 40 to 42° F. The temperature should be lowered slowly."—*A. T. K.*

[80] Hutschenreiter, C. 1936. Das Epiphysan G. Richter in der Hypersexualitäts bekämpfung beim Pferde. (The preparation "epiphysan" of the firm of G. Richter in the treatment of hypersexuality in horses.) *Wiener Tierärztl. Monatsschr.* 23:231-237.

[81] Hucker, G. J., 1937. Mastitis versus The Presence of Mastitis Streptococci in Bovine Mammary Tissue; Tech. Bul. No. 241, New York State Agric. Exp. Sta., Geneva, N. Y.

[82] Dahlberg, A. C., J. J. Kucera, J. C. Hening and G. J. Hucker, 1937. The Composition of Milk as Affected by Latent Mastitis; Tech. Bul. No. 239, New York State Agric. Exp. Sta., Geneva, N. Y.

Book Reviews

I. Small Animal Euthanasia. Report of a Special Committee of the National Veterinary Medical Association of Great Britain and Ireland. 59 pages; 10 plates. Published by the Association, 36 Gordon Square, London, W.C.1. 1937. Price, five shillings.

II. The Veterinary Profession and its Organization for the Control of Animal Diseases. Report of a Committee of Investigation, of the National Veterinary Medical Association of Great Britain and Ireland. 113 pages. Published by the Association, 36 Gordon Square, London, W.C.1. 1937.

III. Handbook on Poultry Diseases. 90 pages; 9 plates; loose-leaf binder. Published by the National Veterinary Medical Association of Great Britain and Ireland. 36 Gordon Square, London, W.C.1. 1937.

The National Veterinary Medical Association of Great Britain and Ireland is ever on the alert to serve its members. Its concern for the special problems of the practitioner has been referred to repeatedly in this magazine. Tangible evidence of its zealous efforts to promote the welfare of British practitioners is the publication of three novel pamphlets on timely subjects for the members of this wide-awake association, and for the benefit of the profession at large.

I. The pamphlet on "Small Animal Euthanasia" is a well written elucidation of the various methods of humane destruction of dogs and cats. Following a short introduction to the objectives of the committee, the lethal drugs in common use (hydrocyanic acid, strychnine, magnesium sulphate) are discussed. The committee concludes that the destruction of dogs by the intravenous injection of solutions of magnesium sulphate is a satisfactory and reliable method. Next the method of prenarcotization by the use of non-volatile anesthetics is taken up. The conclusions are:

1. The degree of narcosis reached in the dog after the subcutaneous injection of morphine sulphate is insufficient to bring into complete subjection the undesirable features of hydrocyanic acid poisoning, and therefore is not recommended.

2. Chloral hydrate either by mouth, intrathoracically, or intraperitoneally cannot be recommended.

3. Nembutal has given the most constant and satisfactory results as a means of inducing general anesthesia in the dog and cat prior to destruction by some other lethal agent.

4. Evipan sodium administered by intravenous injection can be recommended.

5. Avertin is useful in the dog only.

In the discussion of chloroform emphasis is laid on the common misconception of the laity that chloroform in itself is an ideal anesthetic for dogs and cats.

The chapter on lethal chambers is perhaps the most thought-provoking part of the report. The committee concludes that the destruction of dogs by means of chloroform in combination with carbon monoxide falls far short of the ideal, and is unsatisfactory in many respects. Neither motor car exhausts nor coal gas can be recommended as satisfactory means of destruction.

Destruction by means of fire arms, while among the most humane methods is distasteful to many owners. Euthanasia by electric shock will probably become the method of choice, but it needs improvement.

The committee points out that it is not possible to state categorically that any one method of euthanasia is the best. Every case must be governed by its individual requirements. In his choice of a method the operator will always be influenced by a number of factors.

The report deserves to be studied by every practitioner. The newer methods advocated by the committee, and the latest apparatus available are illustrated.

II. The 113-page pamphlet "The Veterinary Profession and its Organization for the Control of Animal Diseases" is a comprehensive report of various aspects of the profession, especially the interrelation of its branches, including frictional relations. Many of the grievances of our British colleagues are remarkably similar to problems of our own. The report, therefore, calls for more than passing notice; its summary and conclusion should make

profitable reading for practitioners and officials in the United States. Perhaps the scope of the report can best be gleaned by random quotation from part I (Causes of friction arising directly or indirectly from recent legislation and other developments).

Where local authorities offer to allow their veterinary officers to perform these after-license inspections at low fees, the committee feels that they are entering into direct competition with practitioners, particularly in those counties where producers are left free to employ their own private veterinary surgeon. Such a practice cannot be approved by the committee.

Some evidence has been received which suggests that certain farmers delay calling in the veterinary practitioner until after a visit from the veterinary officer, in the hope that the latter will advise them about an ailing animal, while other farmers report cases which are not reportable under the Tuberculosis Order. As a result of these practices it is alleged that veterinary practitioners have lost much work since the "Accredited" Herd Scheme began.

It is felt that sending tubes direct to farmers for the collection of blood samples, as was alleged to have been done by a veterinary investigation officer, constitutes a breach of the instructions issued by the Ministry of Agriculture in connection with which it had been made clear that veterinary investigation officers had, in the performance of their duties, to pay due regard to the etiquette of the profession.

It is thought also that veterinary investigation officers should not be utilized by county agricultural organizers as a means of getting free advice for farmers.

The committee feels that members who write for newspapers should refrain from discussing treatment in detail and omit reference to quantities and dosage of drugs owing to the danger of such information being misapplied.

While aware that the Ministry has been issuing bulletins for many years, the committee is of the opinion that greater care should be exercised in describing treatment for farm animals, particularly as such treatment is often improperly carried out by laymen.

The committee feels bound to refer to the very great menace of unqualified practice in the treatment of animals at present allowed by law to continue in this country. Such activities menace the work of the veterinary surgeon both in the town and in the country, and this question, on top of that arising from the chaotic development of veterinary services already referred to, may well become the final blow which will eliminate the veterinary practitioner. The activities referred to are those of quack medicine vendors, travellers of cattle food manufacturers, representatives of large pharmaceutical firms, "quacks", and organizations employing unqualified persons.

One of the greatest sources of irritation in the present chaotic condition of veterinary affairs is the loss of income suffered by practitioners as the result of the haphazard development of local whole-time service.

The committee has noted statements that there has been a diminution in the number of practitioners in some country districts since the development of the whole-time service, and is assured that there are districts where agriculturists are already feeling the dearth of practitioners, but that in spite of these potential openings members are loth to take up general practice.

Part two of the report deals with the present organization of the British veterinary services. Part three forms the summary and the recommendations. The Appendix contains pertinent copies of editorials taken from the *Veterinary Record*."

III. The "Handbook on Poultry Diseases" is a concise pamphlet of 90 pages. It discusses the method of post-mortem examination, specific infectious diseases, parasitic diseases, non-specific diseases, meat inspection, and other government regulations. While decidedly more restricted in scope than the American text book by Barger and Card ("Diseases and Parasites of Poultry"), it possesses the redeeming feature of distribution limited to the veterinary profession and thus avoids the elemental discussion thought proper for laymen by the authors of recent works on the subject of poultry disease.

Among the non-specific diseases mention is made of "mandibular disease", a condition which the reviewer has met on several occasions on poultry farms in the United States. No mention of this disease apparently exists in the American poultry literature. Quoting from the "Handbook":

Affected chicks usually between the ages of two and eight weeks, show necrosis and ulceration with hypertrophy and deformity of either the upper or lower sometimes both beaks. Certain of the mucous glands of these parts appear to become clogged, and this is followed by a local infection with *Actinomyces necrophorus* and the characteristically enlarged, broadened beak follows. This condition is always associated with the feeding of dry mash, usually *ad libitum*. It is curable in many cases simply by substituting wet mash, with or without grain.

How to Breed Dogs, a Popular Exposition of the Scientific Principles Underlying Reproduction and Heredity in Dogs with Special Reference to Their Application. Leon F. Whitney. Cloth. 352 pages. 175 illustrations. $3.50. Orange Judd Publishing Co., New York.

In a straightforward, simple fashion Whitney gives the reader an idea of the complexity of the problems confronting those who breed dogs.

The illustrations throughout the book are excellent and serve well to emphasize and supplement the text. This volume is larger than the author's previous work, "The Basis of Breeding" and is devoted exclusively to dogs. He has used the latest of the scientific researches, chiefly his own, and presents these in chronological order so that the work is given in narrative form, which makes it very understandable and easy to read.

The first part is devoted to Life and the Cell, Reproduction: Cell Division, The Male and the Female Organs of Reproduction—The Mating Cycle—Pregnancy and Influences Affecting Sterility. In this he deals with physiologic fundamentals.

Part Two discusses the principles of heredity, and consideration of how heredity changes and how breeds of dogs may be improved. The manner by which complicated hereditary problems may be studied is indeed a worthy contribution. He blasts many of the most cherished misnomers of animal breeders.

Part Three is a summary of all the studies made up to the present time on specific inheritance in dogs. Coat color inheritance, in most of the popular breeds, inheritance of eye color, coat characteristics, body form, tail characteristics and mental aptitudes are not merely classified for a record, but clarified so that the information can be of definite value to the breeder.

In Part Four Whitney arouses curiosity in his admission that he is now engaged in the creation of several new breeds. His reference to the most popular dog in America, the farm shepherd or barn-yard collie, as a distinctive type and meriting the recognition of kennel associations, strikes a responsive chord in this reviewer.

Especially to be commended are the presentation of the subject, the excellent outline form and the excellent work of the publisher.—*H. J. Mostyn.*

* * * *

Interviewing Animals by Bastian Schmid. The German original, "Begegnung Mit Tieren," was published in Munich in 1935. Translated by Bernard Miall. 223 pages, 60 illustrations. Cloth $3. Houghton-Mifflin Co., Boston.

Doctor Schmid is an animal psychologist who supervises "biological station" of both wild and domestic creatures near Munich, Germany. This work combines the affectionate regard of the animal-lover with the precision of the scientist.

Since childhood, the author has been living with, and acquiring a sympathetic knowledge of the creatures he has observed and studied. The work is primarily for those who, lacking the inclination to read extensively on the subject of animal behaviorism, are willing to accept the observations of others as authoritative.

Doctor Schmid has assembled painstakingly a record of his numerous investigations of the problems of instinct and behavior, such as the mental equipment of birds and animals, their means of communication with their fellows, their psychic interests, their mental images of their individual worlds, the difference between their senses and those of humans, their capacity to learn, and has produced a very excellent book.

"There are myriads of little souls in the animal kingdom, each with its appointed existence, each a world to itself, and yet standing in relation to other little souls in its environment. Is there not something magnificent in this vast alien world? Many of us, alas! have lost all feeling and reverence for it, and we have almost forgotten what wisdom is, in the highest and most primitive sense of the word."

This book is recommended as a spur to the imagination and for a better understanding of animal psychology.—*H. J. Mostyn.*

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